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=> e leonard john p/au  
E1           79     LEONARD JOHN M/AU  
E2           3     LEONARD JOHN N/AU  
E3       106 --> LEONARD JOHN P/AU  
E4           1     LEONARD JOHN PATRICK/AU  
E5           1     LEONARD JOHN PAUL/AU  
E6           2     LEONARD JOHN R/AU  
E7           3     LEONARD JOHN S/AU  
E8           1     LEONARD JOHN T/AU  
E9           1     LEONARD JOHN W JR/AU  
E10          1     LEONARD JOHNATAN N/AU  
E11          1     LEONARD JOHNSON F/AU  
E12          4     LEONARD JON/AU

=> s e3-e5  
L1       108 ("LEONARD JOHN P"/AU OR "LEONARD JOHN PATRICK"/AU OR "LEONARD JOHN PAUL"/AU)

=> e leonard j p/au

E1 1 LEONARD J M R M/AU  
E2 212 LEONARD J N/AU  
E3 355 --> LEONARD J P/AU  
E4 4 LEONARD J P \*/AU  
E5 92 LEONARD J R/AU  
E6 3 LEONARD J R 3RD/AU  
E7 3 LEONARD J R III/AU  
E8 32 LEONARD J S/AU  
E9 2 LEONARD J S JR/AU  
E10 44 LEONARD J T/AU  
E11 741 LEONARD J V/AU  
E12 42 LEONARD J W/AU

=> s e3-e4

L2 359 ("LEONARD J P"/AU OR "LEONARD J P \*/AU)

=> e goldman samuel/au

E1 2 GOLDMAN S Z/AU  
E2 1 GOLDMAN SAM/AU  
E3 13 --> GOLDMAN SAMUEL/AU  
E4 6 GOLDMAN SAMUEL C/AU  
E5 1 GOLDMAN SAMUEL D/AU  
E6 50 GOLDMAN SAMUEL J/AU  
E7 1 GOLDMAN SAMUEL JAY/AU  
E8 1 GOLDMAN SAMUEL L/AU  
E9 5 GOLDMAN SAMUEL M/AU  
E10 1 GOLDMAN SANDY/AU  
E11 6 GOLDMAN SARAH/AU  
E12 2 GOLDMAN SARAH A/AU

=> s e2-e9

L3 78 ("GOLDMAN SAM"/AU OR "GOLDMAN SAMUEL"/AU OR "GOLDMAN SAMUEL C"/AU OR "GOLDMAN SAMUEL D"/AU OR "GOLDMAN SAMUEL J"/AU OR "GOLDMAN SAMUEL JAY"/AU OR "GOLDMAN SAMUEL L"/AU OR "GOLDMAN SAMUEL M"/AU)

=> e goldman s/au

E1 3 GOLDMAN RUTH/AU  
E2 1 GOLDMAN RUTH E/AU  
E3 1458 --> GOLDMAN S/AU  
E4 245 GOLDMAN S A/AU  
E5 3 GOLDMAN S A \*/AU  
E6 56 GOLDMAN S B/AU  
E7 20 GOLDMAN S C/AU  
E8 2 GOLDMAN S D/AU  
E9 24 GOLDMAN S E/AU  
E10 15 GOLDMAN S F/AU  
E11 9 GOLDMAN S G/AU  
E12 11 GOLDMAN S H/AU

=> s e3

L4 1458 "GOLDMAN S"/AU

=> e ohara richard/au

E1 1 OHARA REIJI/AU  
E2 19 OHARA REIKO/AU  
E3 0 --> OHARA RICHARD/AU  
E4 1 OHARA RIE/AU  
E5 1 OHARA RIEKO/AU  
E6 99 OHARA RIICHIRO/AU  
E7 1 OHARA RIKURO/AU  
E8 1 OHARA RURI/AU

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E9      3      OHARA RYO/AU  
E10     13     OHARA RYOICHI/AU  
E11      1     OHARA RYOUICHI/AU  
E12      2     OHARA RYUICHI/AU
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=> e o'hara richard/au  
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=> e ohara r/au  
E1      1      OHARA PATRICK J/AU  
E2      19     OHARA PETER T/AU  
E3      70 --> OHARA R/AU  
E4      1      OHARA R B/AU  
E5      1      OHARA R D/AU  
E6      4      OHARA R J/AU  
E7      1      OHARA R P/AU  
E8      1      OHARA R S/AU  
E9      1      OHARA REIJI/AU  
E10     19     OHARA REIKO/AU  
E11     1      OHARA RIE/AU  
E12     1      OHARA RIEKO/AU
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=> s e3  
L5      70 "OHARA R"/AU
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=> s 11-15  
L6      2059 (L1 OR L2 OR L3 OR L4 OR L5)
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=> s 16 and (IL-12 or RA or arthritis)  
L7      109 L6 AND (IL-12 OR RA OR ARTHRITIS)
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=> s 17 and (antibod? or antagonist?)  
L8      43 L7 AND (ANTIBOD? OR ANTAGONIST?)
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=> dup rem 18  
PROCESSING COMPLETED FOR L8  
L9      20 DUP REM L8 (23 DUPLICATES REMOVED)
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=> d bib ab 1-20
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L9      ANSWER 1 OF 20 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE  
1  
AN      2002:166945 BIOSIS  
DN      PREV200200166945  
TI      Use of IL-12 and IL-12  
       antagonists in the treatment of autoimmune diseases.  
AU      Leonard, John (1); Goldman, Samuel; O'Hara, Richard, Jr.  
CS      (1) Auburn, NH USA
```

ASSIGNEE: Genetics Institute, Inc.  
PI US 6338848 January 15, 2002  
SO Official Gazette of the United States Patent and Trademark Office Patents,  
(Jan. 15, 2002) Vol. 1254, No. 3, pp. No Pagination.  
<http://www.uspto.gov/web/menu/patdata.html>. e-file.  
ISSN: 0098-1133.

DT Patent  
LA English  
AB Method of treating autoimmune conditions are disclosed comprising administering to a mammalian subject **IL-12** or an **IL-12 antagonist**. In certain preferred embodiments the autoimmune condition is one which is promoted by an increase in levels of IFN-gamma or TNF-alpha. Suitable conditions for treatment include multiple sclerosis, systemic lupus erythematosus, rheumatoid **arthritis**, autoimmune pulmonary inflammation, Guillain-Barre syndrome, autoimmune thyroiditis, insulin dependent diabetes melitis and autoimmune inflammatory eye disease.

L9 ANSWER 2 OF 20 WPIDS (C) 2002 THOMSON DERWENT  
AN 2002-147853 [19] WPIDS  
DNC C2002-045892  
TI Composition for modulating immune response, comprises a spore system having a spore and polypeptide, carbohydrate or nucleotide sequence having anti-pathogenic activity.  
DC B04 D16  
IN **GOLDMAN, S; LATHROP, S J; LONGCHAMP, P F; WHALEN, R G**  
PA (MAXY-N) MAXYGEN INC  
CYC 96  
PI WO 2002000232 A2 20020103 (200219)\* EN 137p  
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ  
NL OA PT SD SE SL SZ TR TZ UG ZW  
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK  
DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR  
KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU  
SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW  
AU 2001073009 A 20020108 (200235)  
ADT WO 2002000232 A2 WO 2001-US20372 20010626; AU 2001073009 A AU 2001-73009  
20010626  
FDT AU 2001073009 A Based on WO 200200232  
PRAI US 2000-214161P 20000626  
AB WO 200200232 A UPAB: 20020321  
NOVELTY - A composition (I), comprising a spore system (II) having a spore and a peptide, polypeptide, protein, carbohydrate or nucleotide sequence having anti-pathogenic activity displayed on, bound to or contained within, the spore, is new.  
DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:  
(1) releasing a spore system of interest, comprising:  
(a) transforming a cell capable of sporulation with an exogenous nucleic acid;  
(b) inducing sporulation of the cell, where at least one spore system is produced; and  
(c) lysing the cell to release the spore system;  
(2) displaying a polypeptide at one or more sites of interest on a surface of a spore, comprising:  
(a) transforming a cell capable of sporulation with a recombinant nucleic acid vector, comprising a nucleic acid encoding a polypeptide fused in frame to a nucleic acid encoding a spore protein; and  
(b) expressing a fusion protein comprising the polypeptide and the spore coat protein so that the fusion protein is attached to the spore coat of the spore at one or more site of interest on the spore surface;  
(3) a detection system (DS) comprising (II) which comprises a moiety

that provides a detectable signal and a polypeptide capable of capturing a detectable compound;

(4) delivery of a polypeptide of interest, comprising:

(a) transforming a cell that is capable of sporulating with a nucleic acid encoding the polypeptide;

(b) inducing sporulation of the cell to form a spore; and

(c) delivering the spore to a site of interest;

(5) modulation of an adjuvant effect in an organism, comprising:

(a) generating a non-viable spore, having an adjuvant effect;

(b) isolating the spore; and

(c) contacting the organism with the spore and a nucleic acid, polypeptide, or peptide; and

(6) enhancing (M1) an immune response to an immunogenic polypeptide or peptide in a subject, comprising administering (I).

ACTIVITY - Antibacterial; virucide; anti-HIV (human immunodeficiency virus); cytostatic; neuroprotective; nootropic; hepatotropic; antipyretic; antiinflammatory; antirheumatic; antiarthritic; antidiabetic; immunosuppressive; antipsoriatic; antiallergic; antiasthmatic.

MECHANISM OF ACTION - Modulator of immune response (claimed); vaccine.

Spores from *Bacillus subtilis* were tested to determine if the spores had an adjuvant effect. The specific immunological response of mice to spores and V-antigen mixed together was compared to the specific immunological response of mice to the V-antigen protein alone. 1 micro g, 0.5 micro g or 0.25 micro g of purified recombinant V-antigen was mixed with 5 multiply 10<sup>8</sup> non-recombinant *B. subtilis* spores or used alone. The three V-antigen protein/spore mixtures and three amounts of V-antigen protein were injected intraperitoneally into separate groups of mice, at days 1, 21 and 35. Mice were bled on days 10, 21 and 45. Serum was analyzed for specific and V-antigen immunoglobulins by an indirect enzyme linked immunosorbent assay (ELISA) using standard procedures. The presence of spores in the inoculum increased the **antibody** titer between 10-fold and 1000-fold, depending on the amount of protein inoculated. The data suggested that spores act to augment a specific immune response to immunogenic polypeptide, such as V-antigen protein.

USE - (I) is useful for modulating (producing or enhancing) an immune response of an organism, and for generating a desired product. DS is useful for detecting a compound. (All claimed). The spores are useful in production, packaging, delivering and presentation systems for industrial biocatalyst and in medical applications including immunization and vaccination. The spores are also useful as therapeutics and/or prophylactic agents, and as vaccines against a broad spectrum of immunogens and bacterial, viral and parasitic pathogens and toxins. The spores are also useful for production and immobilization of enzymes and proteins for industrial use, and in a variety of biotechnology settings as carriers for nucleic acids and biotin linked ligands. (II) is useful as sensor and detector. (II) is useful as a vaccine or immunomodulatory agent against a disease or disease causing pathogen including *Staphylococcus* sp., *Streptococcus* sp., viral encephalitis, human immunodeficiency virus (HIV), cytomegalovirus, poliomyelitis, rabies, cancer, typhoid, parasites, anthrax, foot and mouth disease, Alzheimer's disease, hepatitis, diphtheria, pertussis, hemorrhagic fevers, influenza, cholera, meningitis, measles, mumps, Lyme disease, tetanus, yellow fever and pneumonia. (I) is also useful for treating allergy, asthma, autoimmune diseases, e.g. rheumatoid **arthritis**, diabetes mellitus and multiple sclerosis, septic shock, organ transplantation and inflammatory conditions including inflammatory bowel syndrome, psoriasis, pancreatitis, and other immunodeficiencies.

Dwg.0/12

AN 2002:291341 BIOSIS  
DN PREV200200291341  
TI Expression and regulation of the PD-L1 immunoinhibitory molecule on microvascular endothelial cells.  
AU Eppihimer, Michael J. (1); Gunn, Jason; Freeman, Gordon J.; Greenfield, Edward A.; Chernova, Tetyana; Erickson, Jamie; **Leonard, John P.**  
CS (1) Discovery Research: Respiratory Diseases, Wyeth/Genetics Institute, Inc., One Burtt Road, Andover, MA, 01810: meppihimer@genetics.com USA  
SO Microcirculation (New York), (April, 2002) Vol. 9, No. 2, pp. 133-145. <http://www.naturesj.com/mn/index.html>. print.  
ISSN: 1073-9688.  
DT Article  
LA English  
AB Objective: To evaluate the expression and regulation of a novel B7-like protein, PD-L1, the ligand for the immunoinhibitory receptor PD-1 expressed on activated T-cells, on microvascular endothelial cells (ECs). Methods: PD-L1 expression on ECs in vitro and in vivo was quantified by using a dual radiolabeled antibody technique after treatment with interferons (IFN) and IL-12, respectively. Changes in the level of PD-L1 mRNA were determined by using RT-PCR. Results: PD-L1 was observed to be present on ECs under basal conditions. Treatment of ECs with IFN-alpha, -beta and -gamma, but not LPS, was observed to induce elevations in the mRNA and surface expression of PD-L1 on ECs. By using a dual radiolabeled monoclonal antibody (mAb) technique, PD-L1 expression in various tissues of control and IL-12 challenged wild-type and IFN-gamma-deficient mice was measured. A significant increase in PD-L1 expression was observed in tissues at 24 hours after IL-12-challenge, with peak levels of PD-L1 occurring 72 hours after IL-12 challenge. IL-12 was not effective at inducing PD-L1 expression in tissues of IFN-gamma-deficient mice. Conclusions: These data show the expression of a novel B7-like molecule on murine ECs that is mediated by IFN-alpha, -beta, and -gamma, and suggest a potential pathway by which ECs may modulate T-cell function.

L9 ANSWER 4 OF 20 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.  
AN 2001274332 EMBASE  
TI Interleukin-12 gene therapy vaccines: Directing the immune system against minimal residual leukemia.  
AU Dunussi-Joannopoulos K.; **Leonard J.P.**  
CS Dr. K. Dunussi-Joannopoulos, Genetics Institute, One Burtt Road, Andover, MA 01810, United States  
SO Leukemia and Lymphoma, (2001) 41/5-6 (483-492).  
Refs: 60  
ISSN: 1042-8194 CODEN: LELYEA  
CY United Kingdom  
DT Journal; General Review  
FS 016 Cancer  
022 Human Genetics  
025 Hematology  
026 Immunology, Serology and Transplantation  
030 Pharmacology  
037 Drug Literature Index  
LA English  
SL English  
AB Current overall survival rates for patients with AML remain poor and there is need for novel therapeutic approaches. One such approach is to use the patient's own immune system to eliminate minimal residual disease. Recent advances in genetic manipulation of tumor cells, together with a better understanding of the immune mechanisms controlling the host-tumor relationship have led to a flurry of preclinical and clinical studies on tumor cell vaccines. Here we present a brief overview of genetic

manipulation of tumor cells, and highlight important principles of cancer immunity and cancer vaccines. Special emphasis is given on recent work on the role of interleukin-12 (**IL-12**) based vaccines in murine AML. These studies have shown that vaccines with AML cells, genetically modified to secrete **IL-12**, are potent stimulators of the immune system and lead to the development of both prophylactic and therapeutic anti-leukemia immunity.

L9 ANSWER 5 OF 20 WPIDS (C) 2002 THOMSON DERWENT DUPLICATE 3  
AN 2000-524532 [47] WPIDS  
DNN N2000-387705 DNC C2000-155840  
TI Humanized immunoglobulin having a binding specificity to B7-1 (derived from ATCC PTA-263), or B7-2 (derived from ATCC CRL-12524) molecules, modulates immune responses and can therefore treat e.g. autoimmune diseases, infectious diseases.  
DC B04 D16 S03  
IN CARRENO, B; CELNIKER, A C; CO, M S; COLLINS, M; FRIEDRICH, S; GOLDMAN, S; GRAY, G S; KNIGHT, A; OHARA, D; RUP, B; VASQUEZ, M; VELDMAN, G M; WARNER, G; GARVIN, W; GRAY, C S; STUART, F; O'HARA, D  
PA (GEMY) GENETICS INST INC  
CYC 91  
PI WO 2000047625 A2 20000817 (200047)\* EN 158p  
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL  
OA PT SD SE SL SZ TZ UG ZW  
W: AE AL AM AT AU AZ BA BB BG BR BY CA CH CN CR CU CZ DE DK DM EE ES  
FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS  
LT LU LV MA MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL  
TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW  
AU 2000039988 A 20000829 (200062)  
NO 2001003911 A 20011010 (200174)  
EP 1159300 A2 20011205 (200203) EN  
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT  
RO SE SI  
CZ 2001002925 A3 20020116 (200215)  
BR 2000008209 A 20020219 (200222)  
ADT WO 2000047625 A2 WO 2000-US3303 20000209; AU 2000039988 A AU 2000-39988  
20000209; NO 2001003911 A WO 2000-US3303 20000209, NO 2001-3911 20010810;  
EP 1159300 A2 EP 2000-919275 20000209, WO 2000-US3303 20000209; CZ  
2001002925 A3 WO 2000-US3303 20000209, CZ 2001-2925 20000209; BR  
2000008209 A BR 2000-8209 20000209, WO 2000-US3303 20000209  
FDT AU 2000039988 A Based on WO 200047625; EP 1159300 A2 Based on WO  
200047625; CZ 2001002925 A3 Based on WO 200047625; BR 2000008209 A Based  
on WO 200047625  
PRAI US 1999-339596 19990624; US 1999-249011 19990212  
AB WO 200047625 A UPAB: 20000925  
NOVELTY - Humanized immunoglobulin having a binding specificity to B7-1 (derived from ATCC PTA-263), or B7-2 (derived from ATCC CRL-12524) molecules, comprising an antigen binding region of non-human origin and a portion of a human immunoglobulin, is new.  
DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:  
(1) a host cell comprising nucleic acid that encodes a humanized B7-1 **antibody** and/or a humanized B7-2 **antibody**;  
(2) a humanized immunoglobulin light/heavy chain having binding specificity for B7-1 comprising CDR1, CDR2, and CDR3 of the light/heavy chain of murine 1F1 **antibody** and a human light/heavy chain framework region;  
(3) an isolated nucleic acid (N1) comprising a defined 390 base pair (bp) sequence, encoding a defined 130 amino acid human immunoglobulin light chain variable region (P1) of B7-1 (both given in the specification);  
(4) an isolated nucleic acid (N2) comprising a defined 405 base pair

(bp) sequence, encoding a defined 135 amino acid human immunoglobulin heavy chain variable region (P2) of B7-1 (both given in the specification);

(5) an isolated nucleic acid (N3) comprising a defined 396 base pair (bp) sequence, encoding a defined 132 amino acid human immunoglobulin light chain variable region (P3) of B7-2 (both given in the specification);

(6) an isolated nucleic acid (N4) comprising a defined 405 base pair (bp) sequence, encoding a defined 135 amino acid human immunoglobulin heavy chain variable region (P4) of B7-2 (both given in the specification);

(7) a fused gene encoding humanized immunoglobulin light or heavy chain comprising a first nucleic acid sequence encoding an antigen binding region derived from murine 1F1 or 3D1 monoclonal **antibody** and a second nucleic acid sequence encoding a portion of a constant region of an immunoglobulin of human origin;

(8) a method for inhibiting the interaction of a first cell bearing a B7-1 receptor with a second cell bearing B7-1, comprising contacting the first cell with a humanized immunoglobulin having a binding specificity to B7-1, or B7-2 molecules;

(9) a method for treating an individual having a transplanted organ, tissue or cell comprising administering humanized immunoglobulin having a binding specificity to B7-1, or B7-2 molecules;

(10) a method for treating a disease modulated by B7-1 or B7-2;

(11) a method for making a humanized immunoglobulin having binding specificity for B7-1 or B7-2 comprising:

- (a) determining the complementarity determining regions (CDRs) of an **antibody** of non-human origin which has binding specificity for B7-1 or B7-2;
- (b) obtaining a human **antibody** having a framework region amino acid sequence suitable for grafting of the CDRs in (a); and
- (c) grafting the CDRs of (a) with those of (b);

(12) a method for determining the presence or absence of B7-1 or B7-2 in a sample comprising:

- (a) contacting the sample with an **antibody** specific to B7-1 or B7-2 to allow complex formation; and
- (b) detecting the presence or absence of the complex;

(13) a humanized immunoglobulin light or heavy chain having binding specificity for B7-2 comprising CDR1, CDR2, and CDR3 of the light chain of murine 3D1 **antibody**, and a human light or heavy chain framework region;

(14) a method for transplanting cells into an individual comprising:

- (a) obtaining cells from a donor;
- (b) contacting the cells with an immunoglobulin specific to B7-1 and B7-2 and recipient cells from the individual to allow tolerance reduction; and
- (c) introducing the mixture to the individual;

(15) a method for treating a disorder selected from autoimmune diseases, infectious diseases, inflammatory disorders, systemic lupus erythematosus, diabetes mellitus, insulitis, asthma, **arthritis**, inflammatory bowel disease, inflammatory dermatitis, and multiple sclerosis comprising administering a humanized immunoglobulin to B7-1 and B7-2

(16) a method for treating a transplant recipient or preventing transplant rejection in a transplant recipient, comprising administering an immunoglobulin specific to B7-1 and B7-2; and

(17) a method for decreasing an **antibody** response to an antigen in a mammal comprising administering a humanized immunoglobulin specific to B7-1 or B7-2.

ACTIVITY - Immunosuppressive; antiinfective; antiinflammatory; dermatological; antidiabetic; antiasthmatic; antiarthritic; cytostatic; antianemic; neuroprotective.

MECHANISM OF ACTION - Modulation of immune responses; inhibition of T cell costimulation.

Isolated CD28+ T cells were washed once and resuspended in RPMI (not defined) complete medium, supplemented with 2 ng/ml PMA (not defined), to a cell density of 5 multiply 10<sup>5</sup> cells/ml. The CD28+ T cells were added to the antibody/CHO/hB7-2 mixture, incubated for 3 days at 37 deg. C, 5% CO<sub>2</sub>, and T cell proliferation was measured by pulsing for the last 6 hours of culture with 1 uCi of (3H)-thymidine. The cells were harvested on a filter and the incorporated radioactivity was measured in a scintillation counter. Results showed that both antibodies exhibited dose dependent inhibition of B7-2 driven T cell proliferation with similar IC<sub>50</sub> (inhibitory concentration 50%) values of 72 pm (murine anti-hB7-2) and 50 pm (humanized anti-hB7-2) indicating that both antibodies were similar and very effective in inhibiting the B7-2 T cell stimulatory signal. This demonstrated that the high affinity anti-B7-2 mAbs could block B7-2 functionality by inhibiting the activation and/or proliferation of human T cells.

USE - The humanized immunoglobulin with binding specificity to B7-1 and/or B7-2 is useful for treating autoimmune diseases, infectious diseases, inflammatory disorders, systemic lupus erythematosus, diabetes mellitus, insulitis, asthma, arthritis, inflammatory bowel disease, inflammatory dermatitis, and multiple sclerosis. The immunoglobulins are also useful for treating a transplant recipient or preventing transplant rejection in a transplant recipient, and treating proliferative disease (leukemia, lymphoma and cancer), anemia (sickle-cell anemia, thalassemia and aplastic anemia), inborn errors of metabolism, congenital immunodeficiency diseases, and myeloid dysplasia syndrome.

Dwg.0/28

L9 ANSWER 6 OF 20 WPIDS (C) 2002 THOMSON DERWENT DUPLICATE 4  
AN 2000-430395 [37] WPIDS  
CR 1997-132638 [12]; 1997-165283 [15]; 2000-282570 [23]  
DNC C2000-130765  
TI New polynucleotides encoding human cytotoxic T-lymphocyte antigen (CTLA)-8 proteins useful for treating e.g. autoimmune diseases, inflammatory diseases, and microbial infections.  
DC B04 D16  
IN CARLIN, M; GIANNOTTI, J; GOLDEN-FLEET, M M; GOLDMAN, S; JACOBS, K; KELLEHER, K; MI, S; NEBEN, S; PITTMAN, D  
PA (GEMY) GENETICS INST INC  
CYC 1  
PI US 6074849 A 20000613 (200037)\* 25p  
ADT US 6074849 A Provisional US 1995-35347P 19950719, CIP of US 1995-514014 19950811, US 1996-685239 19960718  
PRAI US 1995-35347P 19950719; US 1995-514014 19950811; US 1996-685239 19960718  
AB US 6074849 A UPAB: 20010711  
NOVELTY - An isolated polynucleotide (I) comprising nucleotides 146-544 of an 813 base pair sequence, fully defined in the specification, or its variant resulting from degeneracy of the genetic code, is new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

- (1) a host cell transformed with (I) operably linked to an expression control sequence; and
- (2) producing a human cytotoxic T-lymphocyte antigen (CTLA)-8 protein comprising culturing a host cell of (1), and purifying the human CTLA-8 protein from the culture.

ACTIVITY - Immunosuppressive; neuroprotective; dermatological; antirheumatic; antiarthritic; antithyroid; antidiabetic; ophthalmological; anti-HIV; antibacterial; hepatotropic; anti-inflammatory; virucide; antifungal; protozoacide; antiallergic; antianemic; cytostatic.

MECHANISM OF ACTION - Gene therapy; Interferon production inducer;

Interleukin (IL)-3 inducer; granulocyte-monocyte colony stimulating factor inducer; chemotaxis-stimulator. MRC5 cells were incubated in the presence of human CTLA-8 (B18), and production of IL-6 and IL-8 were measured. Herpes CTLA-8 (IL-7) was used as positive control. B18 showed titerable production of both IL-6 and IL-8.

USE - (I) is useful for treating autoimmune disorders (e.g. multiple sclerosis, systemic lupus erythematosus, rheumatoid **arthritis**, autoimmune thyroiditis, insulin dependent diabetes mellitus, myasthenia gravis, and autoimmune inflammatory eye disease), viral (e.g. human immunodeficiency virus (HIV), hepatitis, herpes, influenza, cytomegalovirus (CMV)), bacterial, fungal (e.g. Candida) or protozoan (e.g. malaria, leishmaniasis) infections. (I) can also be used for treating allergic reactions, and to suppress chronic or acute inflammation associated with infection such as septic shock or systemic inflammatory response syndrome, inflammatory bowel disease, Crohn's disease or resulting from over production cytokines, regulation of hematopoiesis and consequently in the treatment of myeloid or lymphoid cell deficiencies, supports the growth and proliferation of erythroid progenitor cells alone or in combination with other cytokines. (I) can be used in treating anemia, in chemotherapy to treat or prevent consequent myeloid suppression, in treating various stem cell disorders (e.g. aplastic anemia and paroxysmal nocturnal hemoglobinuria), and in repopulating the stem cell compartment post irradiation/chemotherapy, either in vivo or ex vivo as normal cells or genetically manipulated for gene therapy. Human CTLA-8 proteins may be used to inhibit growth and proliferation of vascular endothelial cells, hence effective in inhibiting angiogenesis as well as in treating tumors, as well as to immunize animals to obtain **antibodies** which specifically react with the CTLA-8 protein, and which may inhibit CTLA-8 binding to its receptor.

Dwg.0/10

L9 ANSWER 7 OF 20 WPIDS (C) 2002 THOMSON DERWENT DUPLICATE 5  
AN 2000-282570 [24] WPIDS  
CR 1997-132638 [12]; 1997-165283 [15]; 2000-430395 [36]  
DNC C2000-085202  
TI Novel human CTLA-8 protein useful for treating immunodeficiencies and disorders, in regulating growth, proliferation and/or activity of T and/or B lymphocytes and multiple sclerosis, rheumatoid **arthritis**.  
DC B04 D16  
IN CARLIN, M; GIANNOTTI, J; GOLDEN-FLEET, M M; **GOLDMAN, S**; JACOBS, K; KELLEHER, K; MI, S; NEBEN, S; PITTMAN, D  
PA (GEMY) GENETICS INST INC  
CYC 1  
PI US 6043344 A 20000328 (200024)\* 25p  
ADT US 6043344 A Provisional US 1995-35347P 19950719, CIP of US 1995-504032 19950719, CIP of US 1995-514014 19950811, Div ex US 1996-685239 19960718, US 1998-34810 19980304  
FDT US 6043344 A CIP of US 5707829  
PRAI US 1995-35347P 19950719; US 1995-504032 19950719; US 1995-514014 19950811; US 1996-685239 19960718; US 1998-34810 19980304  
AB US 6043344 A UPAB: 20010711  
NOVELTY - An isolated human CTLA-8 (B18) (I) with a fully defined sequence as given in the specification, is new.

DETAILED DESCRIPTION - (I) comprises a fully defined sequence of 163 (2) amino acids, amino acids 11-, 29- or 31-163 of (2), the fragment of (2) comprising amino acids 11-, 29- or 31-163 of (2), as given in the specification.

An INDEPENDENT CLAIM is also included for the preparation of (I).

ACTIVITY - Immunosuppressive; antiarthritic; antiinflammatory; immunostimulant; antidiabetic; neuroprotective; dermatological; antianemic; antiallergic; antithyroid; antiasthmatic; antibacterial; cytostatic.

MECHANISM OF ACTION - Angiogenesis inhibitor; hematopoiesis regulator; growth or proliferation of vascular endothelial cells inhibitor; tumor growth inhibitor; myeloid, lymphoid cells or their progenitors proliferator; IFN- gamma , IL-3, GM-CSF production inducer; gene therapy. The ability of (I) to inhibit angiogenesis was examined in an angiostatic activity assay. Primary human umbilical cells (HUVECs) were seeded to 2 multiply 10<sup>3</sup> cells/well of a 96 well plate and incubated. The cells were then starved in M199 medium. Conditioned media containing B18 was obtained from transfected COS or stably expressing CHO cells and 1:10, 1:50, 1:250 and 1:1250 were prepared in M199-CS medium containing 100 ng/ml FGF. The dilutions of B18 were added to the starved cells and incubated for 72 hr at 37 deg. C. The cells were then radiolabeled and trypsinized for liquid scintillation counting, after washing. Results showed that human CTLA-8 (B18) inhibits angiogenesis.

USE - (I) is used for treating immune deficiencies and disorders (including severe combined immunodeficiency (SCID), e.g. in regulating growth and proliferation of T and/or B lymphocytes, and effecting the cytolytic activity of NK cells and other cell populations. These immune deficiencies may be genetic or caused by viruses, bacterial or fungal infections. The proteins are also used for boosting the immune system for treating cancer and in the treatment of autoimmune diseases such as multiple sclerosis, systemic lupus erythematosus, rheumatoid **arthritis**, autoimmune pulmonary inflammation, Guillain-Barre syndrome, autoimmune thyroiditis, insulin dependent diabetes mellitus, myasthenia gravis, graft-versus-host disease and autoimmune inflammatory eye disease. They are also used for treating asthma, allergic reactions or other respiratory problems and suppressing chronic or acute inflammation associated with infection such as septic shock or systemic inflammatory response syndrome (SIRS), inflammatory bowel disease and Crohn's disease. (I) is also used for regulating hematopoiesis and consequently in the treatment of myeloid or lymphoid deficiencies i.e. by supporting the growth and proliferation of erythroid progenitor cells, myeloid cells, megakaryocytes, hematopoietic stem cells and thus used for treating anemia, thrombocytopenia, aplastic anemia and paroxysmal nocturnal hemoglobinuria. They also inhibit the growth and proliferation of vascular endothelial cells and thus are effective in inhibiting angiogenesis. The polynucleotides encoding (I) can be used in gene therapy. The proteins are used as immunogens to produce polyclonal or monoclonal **antibodies** useful for performing diagnostic assays for CTLA-8.

DESCRIPTION OF DRAWING(S) - The figure shows the data relating to the ability of CTLA-8 to inhibit angiogenesis.

Dwg.3/7

L9 ANSWER 8 OF 20 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.  
AN 2001:39565 BIOSIS  
DN PREV200100039565  
TI Myelin oligodendrocyte glycoprotein induced EAE in **IL-12**  
p35 deficient mice.  
AU Hunter, S. E. (1); Thibodeaux, D. K. (1); Bouchard, P. (1); **Leonard, J. P. (1)**  
CS (1) Genetics Institute, Inc., Cambridge, MA, 02140 USA  
SO FASEB Journal, (April 20, 2000) Vol. 14, No. 6, pp. A1116. print.  
Meeting Info.: Joint Annual Meeting of the American Association of Immunologists and the Clinical Immunology Society Seattle, Washington, USA  
May 12-16, 2000  
ISSN: 0892-6638.  
DT Conference  
LA English  
SL English  
  
L9 ANSWER 9 OF 20 MEDLINE  
AN 2000021818 MEDLINE

TI Immunological reconstitution and correlation of circulating serum inflammatory mediators/cytokines with the incidence of acute graft-versus-host disease during the first 100 days following unrelated umbilical cord blood transplantation.

AU Abu-Ghosh A.; Goldman S.; Slone V.; Van de Ven C.; Suen Y.; Murphy L.; Sender L.; Cairo M.S.

CS Dr. M.S. Cairo, Georgetown University Medical Center, Lombardi Cancer Center, 2 East Main, 3800 Reservoir Rd. NW, Washington DC 20007, United States

SO Bone Marrow Transplantation, (1999) 24/5 (535-544).  
Refs: 39  
ISSN: 0268-3369 CODEN: BMTRE

CY United Kingdom

DT Journal; Article

FS 005 General Pathology and Pathological Anatomy  
016 Cancer  
025 Hematology  
026 Immunology, Serology and Transplantation  
037 Drug Literature Index

LA English

SL English

AB We investigated early immunological reconstitution and the production of circulating inflammatory mediators and their relationship to aGVHD in children during the first 100 days following unrelated UCBT. Nine patients had an underlying malignant disease (ALL, ANLL), and two, non-malignant diseases (SAA, ALD). The median age was 10 years (range: 1.25-21). Seven of 11 patients were alive by day 100, two died from regimen-related toxicity, and two died from severe aGVHD (grade .gtoreq. III). Myeloid engraftment (ANC .gtoreq. 500 /mm<sup>3</sup> x 2 days) occurred at a median of 24 days (range: 14-55), while platelet engraftment (platelet count .gtoreq. 20,000 /mm<sup>3</sup> untransfused x 7 days) was delayed and occurred at a median of 52 days (range: 33-95). The mean cell dose of CD34+ cells was 3.3 .+-.  
3.51 x 10<sup>5</sup> /kg, and of CD34+/CD41+ cells was 3.94 .+-.  
3.99 x 10<sup>4</sup> /kg. Acute GVHD (grade II-IV) developed in seven patients (77%), and severe aGVHD (grade III-IV) developed in five patients (55%). Serum levels of IL-2R.alpha.; IL-2, IL-4, IL-7, IL-12, and IFN.gamma. were not significantly different between patients with grades 0-I aGVHD and patients with grades II-IV aGVHD. Evaluation of immunological reconstitution on day 90 post UCBT demonstrated an early recovery of the absolute numbers of B cells (CD19+) and NK cells (CD3-/CD56+). Immunoglobulin levels for IgG, IgM and IgA remained normal throughout the study period. PMN functional studies demonstrated normal superoxide generation, bacterial killing (BK), and chemotaxis (CTX). However, both helper (CD3+/CD4+) and suppressor (CD3+/CD8+) T cell subsets remained low during the first 100 days post UCBT with mean .+-.  
s.e.m. values of 120 .+-.  
29 /mm<sup>3</sup> and 10 .+-.  
50 /mm<sup>3</sup>, respectively (normal = 900-2860 /mm<sup>3</sup> (CD3/CD4), normal = 630-1910 /mm<sup>3</sup> (CD3/CD8)). Mitogen response studies showed low blastogenesis to PHA and PWM, with a mean c.p.m. .+-.  
s.e.m. value of 1.7 .+-.  
0.67 x 10<sup>4</sup> for PHA (NL .gtoreq. 75 x 10<sup>3</sup>) and 8.42 .+-.  
4.1 x 10<sup>3</sup> for PWM (NL .gtoreq. 25 x 10<sup>3</sup>). In conclusion, serum levels of inflammatory mediators were not predictive nor did they correlate with the severity of aGVHD. Recovery of NK cells, B cells, and PMN functions occurred within the first 90 days post transplant. However, T cell subsets, CD3+/CD4+ and CD3+/CD8+, and T cell functional activity remained significantly decreased and may account for the high incidence of infectious morbidity seen during this immediate post UCBT period.

L9 ANSWER 12 OF 20 WPIDS (C) 2002 THOMSON DERWENT DUPLICATE 6

AN 1997-132638 [12] WPIDS

CR 1997-165283 [15]; 2000-282570 [23]; 2000-430395 [36]

DNC C1997-042879

TI New nucleic acid encoding the CTLA-8 protein - modulates growth of

DN 20021818 PubMed ID: 10553047  
TI Autocrine regulation of **IL-12** receptor expression is independent of secondary IFN-gamma secretion and not restricted to T and NK cells.  
AU Thibodeaux D K; Hunter S E; Waldburger K E; Bliss J L; Trepicchio W L; Sypek J P; Dunussi-Joannopoulos K; Goldman S J; **Leonard J P**  
CS Preclinical Research and Development, Genetics Institute, Andover, MA 01810, USA.  
SO JOURNAL OF IMMUNOLOGY, (1999 Nov 15) 163 (10) 5257-64.  
Journal code: 2985117R. ISSN: 0022-1767.  
CY United States  
DT Journal; Article; (JOURNAL ARTICLE)  
LA English  
FS Abridged Index Medicus Journals; Priority Journals  
EM 199912  
ED Entered STN: 20000113  
Last Updated on STN: 20000113  
Entered Medline: 19991202  
AB The biological response to **IL-12** is mediated through specific binding to a high affinity receptor complex composed of at least two subunits (designated IL-12Rbeta1 and IL-12Rbeta2) that are expressed on NK cells and activated T cells. The selective loss of IL-12Rbeta2 expression during Th2 T cell differentiation suggests that regulation of this receptor component may govern **IL-12** responsiveness. In murine assays, down-regulation of IL-12Rbeta2 expression can be prevented by treatment with IFN-gamma, indicating that receptor expression and hence **IL-12** responsiveness may be regulated, at least in part, by the local cytokine milieu. In this study, we report that cellular expression of both IL-12Rbeta1 and beta2 mRNA is increased in the lymph nodes of naive mice following systemic administration of murine rIL-12 (rmIL-12). Changes in IL-12R mRNA were associated with increased IFN-gamma secretion following ex vivo activation of lymph node cells with rmIL-12, indicating the presence of a functional receptor complex. Expression of IL-12R mRNA was not restricted to lymph node T cells, and its autocrine regulation was independent of secondary IFN-gamma secretion. Data from fractionated lymph node cells as well as rmIL-12-treated B cell-deficient mice suggest that **IL-12**-responsive B cells may represent an alternative cellular source for IFN-gamma production. However, the strength of the biological response to rmIL-12 is not governed solely by receptor expression, as rmIL-12-induced IFN-gamma secretion from cultured lymph node cells is accessory cell dependent and can be partially blocked by inhibition of B7 costimulation.  
L9 ANSWER 10 OF 20 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.  
AN 1999:275655 BIOSIS  
DN PREV199900275655  
TI Prolonged inhibition of murine lupus by short term therapy with anti-B7 and anti-**IL-12 antibodies** during onset of disease.  
AU Collins, M. (1); Nagle, S. (1); Chung, C. (1); **Goldman, S. (1)**; Sypek, J. (1)  
CS (1) Genetics Institute, Andover, MA, 01810 USA  
SO FASEB Journal, (March 15, 1999) Vol. 13, No. 5 PART 2, pp. A956.  
Meeting Info.: Annual Meeting of the Professional Research Scientists on Experimental Biology 99 Washington, D.C., USA April 17-21, 1999 Federation of American Societies for Experimental Biology  
. ISSN: 0892-6638.  
DT Conference  
LA English  
L9 ANSWER 11 OF 20 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.  
AN 1999315480 EMBASE

vascular endothelial and haematopoietic cells and induces cytokine expression, for treating infection, auto-immune disease, etc..

DC B04 D16

IN CARLIN, M; JACOBS, K; KELLEHER, K; MCCOY, J M; GIANNOTTI, J; GOLDEN-FLEET, M; GOLDMAN, S; MI, S; NEBEN, S; PITTMAN, D; DUCKETT, M C; GOLDEN-FLEET, M M; PITTMAN, D; CARLIN-DUCKETT, M

PA (GEMY) GENETICS INST INC

CYC 23

PI WO 9704097 A2 19970206 (199712)\* EN 50p  
RW: AT BE CH DE DK ES FI FR GB GR IE IT LU MC NL PT SE  
W: AU CA JP MX

AU 9667123 A 19970218 (199723)  
WO 9704097 A3 19970912 (199749)  
US 5707829 A 19980113 (199809) 30p  
EP 839196 A2 19980506 (199822) EN  
R: AT BE CH DE DK ES FI FR GB GR IE IT LI LU MC NL PT SE  
JP 11510045 W 19990907 (199947) 59p  
US 5969093 A 19991019 (199950)  
MX 9800507 A1 19980501 (200007)  
MX 9801120 A1 19990401 (200055)  
AU 727480 B 20001214 (200103)  
AU 727489 B 20001214 (200103)  
AU 2001028001 A 20010517 (200138)#  
AU 2001028002 A 20010802 (200152)#

ADT WO 9704097 A2 WO 1996-US11889 19960718; AU 9667123 A AU 1996-67123 19960218; US 5707829 A US 1995-514014 19950811; EP 839196 A2 EP 1996-927237 19960718, WO 1996-US11889 19960718; JP 11510045 W WO 1996-US11889 19960718, JP 1997-506846 19960718; US 5969093 A Div ex US 1995-514014 19950811, US 1997-833823 19970410; MX 9800507 A1 MX 1998-507 19980116; MX 9801120 A1 MX 1998-1120 19980210; AU 727480 B AU 1996-67123 19960718; AU 727489 B AU 1996-67685 19960808; AU 2001028001 A Div ex AU 1996-67685 19960808, AU 2001-28001 20010314; AU 2001028002 A Div ex AU 1996-67123 19960718, AU 2001-28002 20010314

FDT AU 9667123 A Based on WO 9704097; EP 839196 A2 Based on WO 9704097; JP 11510045 W Based on WO 9704097; AU 727480 B Previous Publ. AU 9667123, Based on WO 9704097; AU 727489 B Previous Publ. AU 9667685, Based on WO 9707198; AU 2001028001 A Div ex AU 727489; AU 2001028002 A Div ex AU 727480

PRAI US 1995-514014 19950811; US 1995-504032 19950719; US 1997-833823 19970410; WO 1996-US12897 19960808; AU 2001-28001 20010314; AU 2001-28002 20010314

AB WO 9704097 A UPAB: 20011001  
A novel isolated polynucleotide (I) comprises: (a) nucleotides (nt) 146-544 of an 813 bp sequence given in the specification; (b) a sequence able to hybridise with (a) or varying from (a) only within the degeneracy of the genetic code; or (c) an allelic variant of (a). Also claimed are: (1) host cells transformed with (I); (2) isolated human CTLA-8 protein which has 163 amino acids (aa), its 11-163, 29-163 or 31-163 regions or any fragments of them with CTLA-8 activity; and (3) antibodies (Ab) which specifically react with CTLA-8 protein.  
USE - (I) encodes proteins with CTLA-8 activity. Treatment of mammals with CTLA-8 (or non-human analogues or IL-17) results in at least one of: (a) inhibition of angiogenesis, growth/proliferation of vascular endothelial cells, tumour cells and angiogenesis-dependent tissue growth; (b) proliferation of myeloid, erythroid or lymphoid cells (or their progeny); or (c) induction of interferon-gamma, IL-3 or GM-CSF prodn (claimed). Opt. CTLA-8 is expressed in vivo from a suitable vector. Typical applications of CTLA-8 are treatment of immune deficiency and disorders requiring modulation of T/B cell growth or proliferation, or of cytolytic natural killer cells, e.g. viral or microbial infection (e.g. HIV, hepatitis, malaria, candidiasis etc.); autoimmune disease (e.g. multiple sclerosis, rheumatoid arthritis, insulin-dependent

diabetes etc.); to boost the immune response in cancer treatment; as antiinflammatories (e.g. in septic shock or Crohn's disease) and in haematopoietic disorders where growth/proliferation of erythroid, myeloid or megakaryocytic cells is needed. Ab can be used to determine CTLA-8, possibly also for treating some tumours or some of the above conditions.  
Dwg.0/7

L9 ANSWER 13 OF 20 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE  
7  
AN 1998:80243 BIOSIS  
DN PREV199800080243  
TI Immunoregulation by interleukin-12 in MB49.1 tumor bearing mice: Cellular and cytokine-mediated effector mechanisms.  
AU Hunter, Sharon E.; Waldburger, Kristine E.; Thibodeaux, Deborah K.; Schaub, Robert G.; **Goldman, Samuel J.**; **Leonard, John P.**  
(1)  
CS (1) Genet. Inst., One Burtt Rd., Andover, MA 01810 USA  
SO European Journal of Immunology, (Dec., 1997) Vol. 27, No. 12, pp. 3438-3446.  
ISSN: 0014-2980.  
DT Article  
LA English  
AB Administration of recombinant murine interleukin (rmIL)-12 to MB49.1 tumor-bearing mice results in dose-dependent regression of the primary tumor and the generation of protective antitumor immunity in the majority of animals. rmIL-12 administration is associated with a marked increase in lymph node cellularity that is predominantly due to the expansion of B220+ B cells as well as CD8+ T cells. Stimulation of lymph node cells from rmIL-12-treated, but not control tumor-bearing mice, with MB49.1 tumor cells in vitro was shown to enhance the secretion of interferon (IFN)-gamma. The magnitude of this in vitro response was dependent on the dose of rmIL-1 2 administered in vivo and mirrored the change in circulating serum IFN-gamma. Furthermore, at the height of the in vitro response to tumor stimulation, the addition of a neutralizing antibody to murine IL-12 suppressed IFN-gamma production, indicating a role for endogenous IL-12 in this antigen-specific cytokine response. Although studies in SCID mice confirmed that an appropriate T cell response was required for rmIL-12-mediated antitumor activity, in immunocompetent animals early tumor regression was not accompanied by cellular infiltration of the tumor. In contrast, a profound increase in tumor-associated inducible nitric oxide synthase (iNOS) was observed in mice receiving rmIL-12 which preceded T cell infiltration of the tumor which could be detected during the second week of IL-12 treatment. Direct tumor killing through the cytotoxic actions of NO via the iNOS pathway may serve as a way of generating tumor antigen which enables the host to mount a subsequent T cell response against the tumor.

L9 ANSWER 14 OF 20 MEDLINE DUPLICATE 8  
AN 1998080712 MEDLINE  
DN 98080712 PubMed ID: 9419442  
TI Regulation of the inflammatory response in animal models of multiple sclerosis by interleukin-12.  
AU **Leonard J P**; Waldburger K E; Schaub R G; Smith T; Hewson A K; Cuzner M L; Goldman S J  
CS Genetics Institute, Andover, MA 01810, USA.  
SO CRITICAL REVIEWS IN IMMUNOLOGY, (1997) 17 (5-6) 545-53. Ref: 54  
Journal code: 8914819. ISSN: 1040-8401.  
CY United States  
DT Journal; Article; (JOURNAL ARTICLE)  
General Review; (REVIEW)  
(REVIEW, TUTORIAL)

LA English  
FS Priority Journals  
EM 199802  
ED Entered STN: 19980306  
Last Updated on STN: 20000303  
Entered Medline: 19980225  
AB Interleukin 12 (**IL-12**), a novel heterodimeric protein produced primarily by antigen-presenting cells, serves as a key regulator of innate and adaptive immune responses. In addition to being a potent inducer of IFN-gamma, **IL-12** is widely considered to be the principal cytokine that regulates the generation of Th1 type effector cells. As the successful induction of experimental autoimmune encephalomyelitis (EAE) is associated with a strong Th1 type cellular response, we have evaluated the role of **IL-12** in regulating the pathogenesis of EAE in SJL/J mice and Lewis rats. In both settings, treatment with **IL-12** was found to accelerate the onset and increase the severity and duration of clinical disease. More importantly, administration of **IL-12** to Lewis rats that had recovered from primary disease was found to trigger clinical relapse. In all instances, **IL-12**-induced exacerbation was associated with a profound increase in iNOS positive macrophages within the perivascular lesions. Although **IL-12**-induced IFN-gamma does not appear to be required for exacerbation of disease, neutralizing **antibodies** against murine **IL-12** delay the onset and reduce the severity of adoptively transferred EAE, indicating a role for endogenous **IL-12** as regulator of disease. Based on the above findings, effective inhibition of **IL-12** *in vivo* may have great therapeutic value in the treatment of MS and other Th1-associated inflammatory disorders.

L9 ANSWER 15 OF 20 MEDLINE DUPLICATE 9  
AN 97118050 MEDLINE  
DN 97118050 PubMed ID: 8958933  
TI Regulation of experimental autoimmune encephalomyelitis by interleukin-12.  
AU Leonard J P; Waldburger K E; Goldman S J  
CS Genetics Institute, Andover, Massachusetts 01810, USA.  
SO ANNALS OF THE NEW YORK ACADEMY OF SCIENCES, (1996 Oct 31) 795 216-26.  
Journal code: 7506858. ISSN: 0077-8923.  
CY United States  
DT Journal; Article; (JOURNAL ARTICLE)  
LA English  
FS Priority Journals; AIDS  
EM 199701  
ED Entered STN: 19970128  
Last Updated on STN: 20000303  
Entered Medline: 19970108  
AB We have evaluated the effects of rmIL-12 on the course of adoptively transferred EAE. When mice were injected with LNC that had been stimulated *in vitro* with PLP in the presence of rmIL-12, the subsequent course of disease was more severe and prolonged than controls. *In vitro* stimulation with PLP in the presence of **IL-12** was associated with an increase in IFN-gamma and decrease in IL-4-producing cells, indicating a preferential expansion of Th1 effector cells. At peak disease, no notable differences in either the cellular composition or cytokine expression within CNS lesions was seen between groups. However, the frequency of macrophages that stained positively for inducible nitric oxide synthase (iNOS) was increased in animals challenged with rmIL-12 treated LNC. These data suggest that in addition to promoting the preferential expansion of IFN-gamma-producing cells by rmIL-12 treatment *in vitro*, *in vivo* effects leading to macrophage activation and iNOS expression may contribute to the severe, protracted course of CNS inflammation in this model. In contrast, treatment of mice with an

**antibody** to murine **IL-12** following cell transfer completely prevented paralysis with only 40% of the mice developing mild disease. These data suggest that endogenous **IL-12** plays a pivotal role in the pathogenesis of this model of autoimmune disease.

L9 ANSWER 16 OF 20 CAPLUS COPYRIGHT 2002 ACS  
AN 1997:285388 CAPLUS  
DN 126:329093  
TI Effects of interleukin 12 on hematopoietic stem and progenitor cells  
AU Neben, Steven; Leonard, John; **Goldman, Samuel**; Ploemacher, Rob E.  
CS Department of Immunology and Hematopoiesis, Genetics Institute, Inc., Cambridge, MA, USA  
SO Bone Marrow Transplantation: Basic and Clinical Studies, [Papers presented at the International Symposium on BMT--Basic and Clinical Studies], Tokyo, Oct. 9-10, 1995 (1996), Meeting Date 1995, 28-35. Editor(s): Ikehara, Susumu; Takaku, Fumimaro; Good, Robert A. Publisher: Springer, Tokyo, Japan.  
CODEN: 64HVAW  
DT Conference; General Review  
LA English  
AB A review with 34 refs. Interleukin-12 (**IL-12**) has been shown to possess potent immunomodulatory activity. It has a unique structure among cytokines, consisting of two covalently linked subunits, one with homol. to other members of the cytokine superfamily, the other being highly homologous to gp130, the signaling subunit of a no. of cytokine receptors. Here we summarize studies showing that **IL-12** is a hematopoietic growth factor with potent activity on hematopoietic stem and progenitor cells. In clonal and liq. culture assays, **IL-12** synergizes with IL-3 and Steel Factor to increase the no. of colonies as well as to expand both stem and progenitor cell content in the cultures. In stroma-dependent long-term bone marrow cultures, **IL-12** addn. causes a decrease in cell prodn. in the first week after inoculation of whole bone marrow cells, followed by an increase in both mature cells and progenitor cells over the next 3 wk. The initial decrease appears to be mediated by **IL-12**-induced prodn. of IFN-.gamma., possibly by natural killer cells and/or T cells which do not persist in these cultures. Studies in naive mice demonstrate a similar acute decrease in peripheral leukocyte count, mediated by IFN-.gamma., upon administration of **IL-12**. In contrast, despite a significant decrease in peripheral platelet count, reticulated platelets become elevated and mean megakaryocyte ploidy in the bone marrow shifts from 16N to 32N during **IL-12** treatment. These **IL-12**-mediated effects on megakaryopoiesis are abrogated by simultaneous treatment of mice with **antibodies** against IFN-.gamma.. These studies provide further information on the potential physiol. role and applications of **IL-12** outside the immune system.

L9 ANSWER 17 OF 20 WPIDS (C) 2002 THOMSON DERWENT DUPLICATE 10  
AN 1995-336810 [43] WPIDS  
DNC C1995-148498  
TI Use of interleukin-I2 or an Il-I2 **antagonist** - for treating autoimmune conditions, eg. multiple sclerosis, lupus, rheumatoid **arthritis** or diabetes.  
DC B04  
IN **GOLDMAN, S; LEONARD, J P; OHARA, R; LEONARD, J; O'HARA, R**  
PA (GEMY) GENETICS INST INC  
CYC 24  
PI WO 9524918 A1 19950921 (199543)\* EN 37p

RW: AT BE CH DE DK ES FR GB GR IE IT LU MC NL PT SE  
W: AU CA JP

AU 9519749 A 19951003 (199602)  
ZA 9500960 A 19951227 (199605) 33p  
EP 750509 A1 19970102 (199706) EN  
R: AT BE CH DE DK ES FR GB GR IE IT LI LU MC NL PT SE  
JP 09510444 W 19971021 (199801) 33p  
AU 689236 B 19980326 (199826)  
IL 112677 A 20000131 (200015)  
TW 400233 A 20000801 (200109)  
US 6338848 B1 20020115 (200208)  
EP 1179348 A2 20020213 (200219) EN  
R: AT BE CH DE DK ES FR GB GR IE IT LI LU MC NL PT SE  
EP 750509 B1 20020515 (200234) EN  
R: AT BE CH DE DK ES FR GB GR IE IT LI LU MC NL PT SE

ADT WO 9524918 A1 WO 1995-US2550 19950307; AU 9519749 A AU 1995-19749  
19950307; ZA 9500960 A ZA 1995-960 19950207; EP 750509 A1 EP 1995-912666  
19950307, WO 1995-US2550 19950307; JP 09510444 W JP 1995-524044 19950307,  
WO 1995-US2550 19950307; AU 689236 B AU 1995-19749 19950307; IL 112677 A  
IL 1995-112677 19950216; TW 400233 A TW 1995-101380 19950214; US 6338848  
B1 Cont of US 1994-212629 19940314, Cont of US 1995-560943 19951120, US  
2000-513380 20000225; EP 1179348 A2 Div ex EP 1995-912666 19950307, EP  
2001-117762 19950307; EP 750509 B1 EP 1995-912666 19950307, WO 1995-US2550  
19950307, Related to EP 2001-117762 19950307

FDT AU 9519749 A Based on WO 9524918; EP 750509 A1 Based on WO 9524918; JP  
09510444 W Based on WO 9524918; AU 689236 B Previous Publ. AU 9519749,  
Based on WO 9524918; EP 1179348 A2 Div ex EP 750509; EP 750509 B1 Related  
to EP 1179348, Based on WO 9524918

PRAI US 1994-212629 19940314; US 1995-560943 19951120; US 2000-513380  
20000225

AB WO 9524918 A UPAB: 19951102  
A method for treating in a mammalian subject an autoimmune condition  
comprises administering (i) an interleukin-I2 (IL-I2) **antagonist**  
or (ii) IL-I2.  
USE - The method is used partic. for autoimmune conditions which are  
promoted by increased levels of TNF-alpha or IFN-gamma (claimed). It can  
be used for treating multiple sclerosis, systemic lupus erythematosus,  
rheumatoid **arthritis**, autoimmune pulmonary inflammation,  
Ciuillan-Barre syndrome, autoimmune thyroiditis, insulin dependent  
diabetes mellitus or autoimmune inflammatory eye disease (claimed).  
Dwg.0/6

L9 ANSWER 18 OF 20 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE  
11

AN 1995:101409 BIOSIS

DN PREV199598115709

TI Prevention of experimental autoimmune encephalomyelitis by  
**antibodies** against interleukin 12.

AU Leonard, J. P. (1); Waldburger, K. E.; Goldman, S. J.

CS (1) Genetics Inst., Preclinical Biol., 87 Cambridge Park Dr., Cambridge,  
MA 02140 USA

SO Journal of Experimental Medicine, (1995) Vol. 181, No. 1, pp. 381-386.  
ISSN: 0022-1007.

DT Article

LA English

AB Experimental allergic encephalomyelitis (EAE) is an autoimmune disease of  
the central nervous system that can be transferred to naive mice via CD4+  
T cells isolated from appropriately immunized mice. We have evaluated the  
effects of recombinant murine interleukin 12 (rmIL-12), a potent inducer  
of interferon gamma (IFN-gamma) and promoter of Th1 T cell development, on  
the course of adoptively transferred EAE. The transfer of lymph node cells  
(LNC) isolated from proteolipid protein (PLP)-primed animals and

stimulated in vitro with PLP to naive mice resulted in a progressive paralytic disease culminating in complete hind limb paralysis in the majority of the recipients. When mice were injected with LNC that had been stimulated in vitro with PLP in the presence of rmIL-12, the subsequent course of disease was more severe and prolonged. The addition of rmIL-12 during the in vitro stimulation with PLP resulted in a 10-fold increase in IFN-gamma and a 2-fold increase in tumor necrosis factor (TNF)  $\alpha$  in the supernatants, relative to LNC stimulated with PLP alone. However, neutralization of IFN-gamma or TNF-alpha in vitro with specific **antibodies** did not abrogate the ability of rmIL-12 to exacerbate the subsequent disease. Similarly, mice treated with rmIL-12 in vivo after the transfer of antigen-stimulated LNC developed a more severe and prolonged course of disease compared with vehicle-treated control animals. In contrast, treatment of mice with an **antibody** to murine **IL-12** after cell transfer completely prevented paralysis, with only 40% of the mice developing mild disease. These results demonstrate that in vitro stimulation of antigen primed LNC with PLP and rmIL-12 enhances their subsequent encephalitogenicity. Furthermore, inhibition of endogenous **IL-12** in vivo after LNC transfer prevented paralysis, suggesting that endogenous **IL-12** plays a pivotal role in the pathogenesis of this model of autoimmune disease.

L9 ANSWER 19 OF 20 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE  
12  
AN 1993:343704 BIOSIS  
DN PREV199396040704  
TI Resolution of cutaneous leishmaniasis: Interleukin 12 initiates a protective T helper type 1 immune response.  
AU Sypek, Joseph P. (1); Chung, Charles L.; Mayor, Sharon E. H.; Subramanyam, Janaki M.; Goldman, Samuel L.; Sieburth, Derek S.; Wolf, Stanley F.; Schaub, Robert G.  
CS (1) Dep. Preclin. Biol., Genetics Inst. Inc., 87 Cambridge Park Dr., Cambridge, MA 02140 USA  
SO Journal of Experimental Medicine, (1993) Vol. 177, No. 6, pp. 1797-1802.  
ISSN: 0022-1007.  
DT Article  
LA English  
AB Resistance to Leishmania major in mice is associated with the appearance of distinct T helper type 1 (Th1) and Th2 subsets. T cells from lymph nodes draining cutaneous lesions of resistant mice are primarily interferon  $\gamma$  (IFN-gamma)-producing Th1 cells. In contrast, T cells from susceptible mice are principally Th2 cells that generate interleukin 4 (IL-4). Although existing evidence is supportive of a role for IFN-gamma in the generation of Th1 cells, additional factors may be required for a protective response to be maintained. A potential candidate is **IL-12**, a heterodimeric cytokine produced by monocytes and B cells that has multiple effects on T and natural killer cell function, including inducing IFN-gamma production. Using an experimental leishmanial model we have observed that daily intraperitoneal administration at the time of parasite challenge of either 0.33  $\mu$ g **IL-12** (a consecutive 5 d/wk for 5 wk) or 1.0  $\mu$ g **IL-12** per mouse (only a consecutive 5 d) caused a gt 75% reduction in parasite burden at the site of infection, in highly susceptible BALB/c mice. Delay of treatment by 1 wk had less of a protective effect. Concomitant with these protective effects was an increase in IFN-gamma and a decrease in IL-4 production, as measured by enzyme-linked immunosorbent assay of supernatants generated from popliteal lymph node cells stimulated with leishmanial antigen in vitro. The reduction in parasite numbers induced by **IL-12** therapy was still apparent at 10 wk postinfection. In addition, we observed that the administration of a rabbit anti-recombinant murine **IL-12** polyclonal

**antibody** (200 mu-g i.p. every other day for 25 d) at the time of infection to resistant C57Bl/6 mice exacerbated disease. These effects were accompanied by a shift in IFN-gamma production in vitro by antigen-stimulated lymph node cells indicative of a Th2-like response. These findings suggest that **IL-12** has an important role in initiating a Th1 response and protective immunity.

L9 ANSWER 20 OF 20 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.  
AN 92231358 EMBASE  
DN 1992231358  
TI Observations, legends, and conjectures concerning restricted T-cell receptor usage and autoimmune disease.  
AU Esch T.; Clark L.; Zhang X.-M.; **Goldman S.**; Heber-Katz E.  
CS Wistar Institute, 3601 Spruce Street, Philadelphia, PA 19104, United States  
SO Critical Reviews in Immunology, (1991) 11/5 (249-264).  
ISSN: 1040-8401 CODEN: CCRIDE  
CY United States  
DT Journal; General Review  
FS 005 General Pathology and Pathological Anatomy  
026 Immunology, Serology and Transplantation  
030 Pharmacology  
037 Drug Literature Index  
LA English  
SL English  
AB It has become clear over the past few years that a variety of experimental autoimmune conditions are mediated by T cells bearing a highly restricted subset of antigen receptors. This restricted TcR usage raises important questions concerning not only the recognition of autoantigens, but also the pathogenic mechanisms underlying many models of autoimmunity. Furthermore, the extension of these findings in certain cases to human disease has raised the possibility of specific therapeutic immune intervention. In this review, we examine the available data on restricted T-cell receptor usage in autoimmune disorders and explore the interpretations and the theoretical and practical implications of these findings.

=> d his

(FILE 'HOME' ENTERED AT 11:40:44 ON 11 JUL 2002)

FILE 'BIOSIS, MEDLINE, AGRICOLA, EMBASE, CABAB, WPIDS, JAPIO, BIOTECHDS, LIFESCI, CAPLUS, USPATFULL, USPAT2' ENTERED AT 11:42:50 ON 11 JUL 2002  
E LEONARD JOHN P/AU

L1 108 S E3-E5  
E LEONARD J P/AU  
L2 359 S E3-E4  
E GOLDMAN SAMUEL/AU  
L3 78 S E2-E9  
E GOLDMAN S/AU  
L4 1458 S E3  
E OHARA RICHARD/AU  
E OHARA R/AU  
L5 70 S E3  
L6 2059 S L1-L5  
L7 109 S L6 AND (IL-12 OR RA OR ARTHRITIS)  
L8 43 S L7 AND (ANTIBOD? OR ANTAGONIST?)  
L9 20 DUP REM L8 (23 DUPLICATES REMOVED)

=> s 19 and p40

L10 1 L9 AND P40

=> d bib ab

L10 ANSWER 1 OF 1 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.  
AN 2001:39565 BIOSIS  
DN PREV200100039565  
TI Myelin oligodendrocyte glycoprotein induced EAE in **IL-12**  
p35 deficient mice.  
AU Hunter, S. E. (1); Thibodeaux, D. K. (1); Bouchard, P. (1); **Leonard,**  
**J. P. (1)**  
CS (1) Genetics Institute, Inc., Cambridge, MA, 02140 USA  
SO FASEB Journal, (April 20, 2000) Vol. 14, No. 6, pp. A1116. print.  
Meeting Info.: Joint Annual Meeting of the American Association of  
Immunologists and the Clinical Immunology Society Seattle, Washington, USA  
May 12-16, 2000  
ISSN: 0892-6638.  
DT Conference  
LA English  
SL English

=> FIL STNGUIDE

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CA SUBSCRIBER PRICE	-0.62	-0.62

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=> s 19 and (fragment? or epitop?)

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LIFESCI, CAPLUS, USPATFULL, USPAT2' ENTERED AT 11:42:50 ON 11 JUL 2002

	E LEONARD JOHN P/AU
L1	108 S E3-E5
	E LEONARD J P/AU
L2	359 S E3-E4
	E GOLDMAN SAMUEL/AU
L3	78 S E2-E9
	E GOLDMAN S/AU
L4	1458 S E3
	E OHARA RICHARD/AU
	E OHARA R/AU
L5	70 S E3
L6	2059 S L1-L5
L7	109 S L6 AND (IL-12 OR RA OR ARTHRITIS)

L8 43 S L7 AND (ANTIBOD? OR ANTAGONIST?)  
L9 20 DUP REM L8 (23 DUPLICATES REMOVED)  
L10 1 S L9 AND P40

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0 "GOLDMAN S "/AU  
0 "OHARA R"/AU  
4 IL  
28 12  
0 IL-12  
(IL(W)12)  
2 RA  
0 ARTHRITIS  
1 ANTIBOD?  
0 ANTAGONIST?  
14 FRAGMENT?  
0 EPITOP?  
L11 0 L8 AND (FRAGMENT? OR EPITOP?)  
  
=> s 19 and (fragment? or epitop?)  
L9 CANNOT BE SEARCHED IN STNGUIDE  
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=> s RA or rheumatoid arthritis

2 RA  
0 RHEUMATOID  
0 ARTHRITIS  
0 RHEUMATOID ARTHRITIS  
(RHEUMATOID(W)ARTHRITIS)  
L12 2 RA OR RHEUMATOID ARTHRITIS

=> file biosis medline agricola embase caba wpids japiro biotechds lifesci caplus uspatall

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CA INDEXING COPYRIGHT (C) 2002 AMERICAN CHEMICAL SOCIETY (ACS)

FILE 'USPAT2' ENTERED AT 11:55:50 ON 11 JUL 2002  
CA INDEXING COPYRIGHT (C) 2002 AMERICAN CHEMICAL SOCIETY (ACS)

=> s 19 and (fragment? or epitop?)  
L13 3 L9 AND (FRAGMENT? OR EPITOP?)

=> d bib ab 1-3

L13 ANSWER 1 OF 3 WPIDS (C) 2002 THOMSON DERWENT  
AN 2002-147853 [19] WPIDS  
DNC C2002-045892

TI Composition for modulating immune response, comprises a spore system having a spore and polypeptide, carbohydrate or nucleotide sequence having anti-pathogenic activity.

DC B04 D16

IN GOLDMAN, S; LATHROP, S J; LONGCHAMP, P F; WHALEN, R G  
PA (MAXY-N) MAXYGEN INC

CYC 96

PI WO 2002000232 A2 20020103 (200219)\* EN 137p

RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ  
NL OA PT SD SE SL SZ TR TZ UG ZW

W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CO CR CU CZ DE DK  
DM DZ EC EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR  
KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU  
SD SE SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW

AU 2001073009 A 20020108 (200235)

ADT WO 2002000232 A2 WO 2001-US20372 20010626; AU 2001073009 A AU 2001-73009  
20010626

FDT AU 2001073009 A Based on WO 200200232

PRAI US 2000-214161P 20000626

AB WO 200200232 A UPAB: 20020321

NOVELTY - A composition (I), comprising a spore system (II) having a spore and a peptide, polypeptide, protein, carbohydrate or nucleotide sequence having anti-pathogenic activity displayed on, bound to or contained within, the spore, is new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

- (1) releasing a spore system of interest, comprising:
  - (a) transforming a cell capable of sporulation with an exogenous nucleic acid;
  - (b) inducing sporulation of the cell, where at least one spore system is produced; and
    - (c) lysing the cell to release the spore system;
    - (2) displaying a polypeptide at one or more sites of interest on a surface of a spore, comprising:
      - (a) transforming a cell capable of sporulation with a recombinant nucleic acid vector, comprising a nucleic acid encoding a polypeptide fused in frame to a nucleic acid encoding a spore protein; and
      - (b) expressing a fusion protein comprising the polypeptide and the spore coat protein so that the fusion protein is attached to the spore coat of the spore at one or more site of interest on the spore surface;
    - (3) a detection system (DS) comprising (II) which comprises a moiety that provides a detectable signal and a polypeptide capable of capturing a detectable compound;
    - (4) delivery of a polypeptide of interest, comprising:
      - (a) transforming a cell that is capable of sporulating with a nucleic acid encoding the polypeptide;
      - (b) inducing sporulation of the cell to form a spore; and
      - (c) delivering the spore to a site of interest;
    - (5) modulation of an adjuvant effect in an organism, comprising:
      - (a) generating a non-viable spore, having an adjuvant effect;
      - (b) isolating the spore; and
      - (c) contacting the organism with the spore and a nucleic acid, polypeptide, or peptide; and
    - (6) enhancing (M1) an immune response to an immunogenic polypeptide or peptide in a subject, comprising administering (I).

ACTIVITY - Antibacterial; virucide; anti-HIV (human immunodeficiency virus); cytostatic; neuroprotective; nootropic; hepatotropic; antipyretic; antiinflammatory; antirheumatic; antiarthritic; antidiabetic; immunosuppressive; antipsoriatic; antiallergic; antiasthmatic.

MECHANISM OF ACTION - Modulator of immune response (claimed); vaccine.

Spores from *Bacillus subtilis* were tested to determine if the spores

had an adjuvant effect. The specific immunological response of mice to spores and V-antigen mixed together was compared to the specific immunological response of mice to the V-antigen protein alone. 1 micro g, 0.5 micro g or 0.25 micro g of purified recombinant V-antigen was mixed with 5 multiply 10<sup>8</sup> non-recombinant *B. subtilis* spores or used alone. The three V-antigen protein/spore mixtures and three amounts of V-antigen protein were injected intraperitoneally into separate groups of mice, at days 1, 21 and 35. Mice were bled on days 10, 21 and 45. Serum was analyzed for specific and V-antigen immunoglobulins by an indirect enzyme linked immunosorbent assay (ELISA) using standard procedures. The presence of spores in the inoculum increased the **antibody titer** between 10-fold and 1000-fold, depending on the amount of protein inoculated. The data suggested that spores act to augment a specific immune response to immunogenic polypeptide, such as V-antigen protein.

USE - (I) is useful for modulating (producing or enhancing) an immune response of an organism, and for generating a desired product. DS is useful for detecting a compound. (All claimed). The spores are useful in production, packaging, delivering and presentation systems for industrial biocatalyst and in medical applications including immunization and vaccination. The spores are also useful as therapeutics and/or prophylactic agents, and as vaccines against a broad spectrum of immunogens and bacterial, viral and parasitic pathogens and toxins. The spores are also useful for production and immobilization of enzymes and proteins for industrial use, and in a variety of biotechnology settings as carriers for nucleic acids and biotin linked ligands. (II) is useful as sensor and detector. (II) is useful as a vaccine or immunomodulatory agent against a disease or disease causing pathogen including *Staphylococcus* sp., *Streptococcus* sp., viral encephalitis, human immunodeficiency virus (HIV), cytomegalovirus, poliomyelitis, rabies, cancer, typhoid, parasites, anthrax, foot and mouth disease, Alzheimer's disease, hepatitis, diphtheria, pertussis, hemorrhagic fevers, influenza, cholera, meningitis, measles, mumps, Lyme disease, tetanus, yellow fever and pneumonia. (I) is also useful for treating allergy, asthma, autoimmune diseases, e.g. rheumatoid **arthritis**, diabetes mellitus and multiple sclerosis, septic shock, organ transplantation and inflammatory conditions including inflammatory bowel syndrome, psoriasis, pancreatitis, and other immunodeficiencies.

Dwg.0/12

L13 ANSWER 2 OF 3 WPIDS (C) 2002 THOMSON DERWENT  
AN 2000-282570 [24] WPIDS  
CR 1997-132638 [12]; 1997-165283 [15]; 2000-430395 [36]  
DNC C2000-085202  
TI Novel human CTLA-8 protein useful for treating immunodeficiencies and disorders, in regulating growth, proliferation and/or activity of T and/or B lymphocytes and multiple sclerosis, rheumatoid **arthritis**.  
DC B04 D16  
IN CARLIN, M; GIANNOTTI, J; GOLDEN-FLEET, M M; GOLDMAN, S; JACOBS, K; KELLEHER, K; MI, S; NEBEN, S; PITTMAN, D  
PA (GEMY) GENETICS INST INC  
CYC 1  
PI US 6043344 A 20000328 (200024)\* 25p  
ADT US 6043344 A Provisional US 1995-35347P 19950719, CIP of US 1995-504032 19950719, CIP of US 1995-514014 19950811, Div ex US 1996-685239 19960718, US 1998-34810 19980304  
FDT US 6043344 A CIP of US 5707829  
PRAI US 1995-35347P 19950719; US 1995-504032 19950719; US 1995-514014 19950811; US 1996-685239 19960718; US 1998-34810 19980304  
AB US 6043344 A UPAB: 20010711  
NOVELTY - An isolated human CTLA-8 (B18) (I) with a fully defined sequence as given in the specification, is new.

DETAILED DESCRIPTION - (I) comprises a fully defined sequence of 163

(2) amino acids, amino acids 11-, 29- or 31-163 of (2), the fragment of (2) comprising amino acids 11-, 29- or 31-163 of (2), as given in the specification.

An INDEPENDENT CLAIM is also included for the preparation of (I).

ACTIVITY - Immunosuppressive; antiarthritic; antiinflammatory; immunostimulant; antidiabetic; neuroprotective; dermatological; antianemic; antiallergic; antithyroid; antiasthmatic; antibacterial; cytotoxic.

MECHANISM OF ACTION - Angiogenesis inhibitor; hematopoiesis regulator; growth or proliferation of vascular endothelial cells inhibitor; tumor growth inhibitor; myeloid, lymphoid cells or their progenitors proliferator; IFN- gamma, IL-3, GM-CSF production inducer; gene therapy. The ability of (I) to inhibit angiogenesis was examined in an angiotropic activity assay. Primary human umbilical cells (HUVECs) were seeded to 2 multiply 10<sup>3</sup> cells/well of a 96 well plate and incubated. The cells were then starved in M199 medium. Conditioned media containing B18 was obtained from transfected COS or stably expressing CHO cells and 1:10, 1:50, 1:250 and 1:1250 were prepared in M199-CS medium containing 100 ng/ml FGF. The dilutions of B18 were added to the starved cells and incubated for 72 hr at 37 deg. C. The cells were then radiolabeled and trypsinized for liquid scintillation counting, after washing. Results showed that human CTLA-8 (B18) inhibits angiogenesis.

USE - (I) is used for treating immune deficiencies and disorders (including severe combined immunodeficiency (SCID), e.g. in regulating growth and proliferation of T and/or B lymphocytes, and effecting the cytolytic activity of NK cells and other cell populations. These immune deficiencies may be genetic or caused by viruses, bacterial or fungal infections. The proteins are also used for boosting the immune system for treating cancer and in the treatment of autoimmune diseases such as multiple sclerosis, systemic lupus erythematosus, rheumatoid arthritis, autoimmune pulmonary inflammation, Guillain-Barre syndrome, autoimmune thyroiditis, insulin dependent diabetes mellitus, myasthenia gravis, graft-versus-host disease and autoimmune inflammatory eye disease. They are also used for treating asthma, allergic reactions or other respiratory problems and suppressing chronic or acute inflammation associated with infection such as septic shock or systemic inflammatory response syndrome (SIRS), inflammatory bowel disease and Crohn's disease. (I) is also used for regulating hematopoiesis and consequently in the treatment of myeloid or lymphoid deficiencies i.e. by supporting the growth and proliferation of erythroid progenitor cells, myeloid cells, megakaryocytes, hematopoietic stem cells and thus used for treating anemia, thrombocytopenia, aplastic anemia and paroxysmal nocturnal hemoglobinuria. They also inhibit the growth and proliferation of vascular endothelial cells and thus are effective in inhibiting angiogenesis. The polynucleotides encoding (I) can be used in gene therapy. The proteins are used as immunogens to produce polyclonal or monoclonal antibodies useful for performing diagnostic assays for CTLA-8.

DESCRIPTION OF DRAWING(S) - The figure shows the data relating to the ability of CTLA-8 to inhibit angiogenesis.

Dwg.3/7

L13 ANSWER 3 OF 3 WPIDS (C) 2002 THOMSON DERWENT

AN 1997-132638 [12] WPIDS

CR 1997-165283 [15]; 2000-282570 [23]; 2000-430395 [36]

DNC C1997-042879

TI New nucleic acid encoding the CTLA-8 protein - modulates growth of vascular endothelial and haematopoietic cells and induces cytokine expression, for treating infection, auto-immune disease, etc..

DC B04 D16

IN CARLIN, M; JACOBS, K; KELLEHER, K; MCCOY, J M; GIANNOTTI, J; GOLDEN-FLEET, M; GOLDMAN, S; MI, S; NEBEN, S; PITTMAN, D; DUCKETT, M C; GOLDEN-FLEET, M M; PITTMAN, D; CARLIN-DUCKETT, M

PA (GEMY) GENETICS INST INC  
CYC 23  
PI WO 9704097 A2 19970206 (199712)\* EN 50p  
RW: AT BE CH DE DK ES FI FR GB GR IE IT LU MC NL PT SE  
W: AU CA JP MX  
AU 9667123 A 19970218 (199723)  
WO 9704097 A3 19970912 (199749)  
US 5707829 A 19980113 (199809) 30p  
EP 839196 A2 19980506 (199822) EN  
R: AT BE CH DE DK ES FI FR GB GR IE IT LI LU MC NL PT SE  
JP 11510045 W 19990907 (199947) 59p  
US 5969093 A 19991019 (199950)  
MX 9800507 A1 19980501 (200007)  
MX 9801120 A1 19990401 (200055)  
AU 727480 B 20001214 (200103)  
AU 727489 B 20001214 (200103)  
AU 2001028001 A 20010517 (200138) #  
AU 2001028002 A 20010802 (200152) #  
ADT WO 9704097 A2 WO 1996-US11889 19960718; AU 9667123 A AU 1996-67123  
19960218; US 5707829 A US 1995-514014 19950811; EP 839196 A2 EP  
1996-927237 19960718, WO 1996-US11889 19960718; JP 11510045 W WO  
1996-US11889 19960718, JP 1997-506846 19960718; US 5969093 A Div ex US  
1995-514014 19950811, US 1997-833823 19970410; MX 9800507 A1 MX 1998-507  
19980116; MX 9801120 A1 MX 1998-1120 19980210; AU 727480 B AU 1996-67123  
19960718; AU 727489 B AU 1996-67685 19960808; AU 2001028001 A Div ex AU  
1996-67685 19960808, AU 2001-28001 20010314; AU 2001028002 A Div ex AU  
1996-67123 19960718, AU 2001-28002 20010314  
FDT AU 9667123 A Based on WO 9704097; EP 839196 A2 Based on WO 9704097; JP  
11510045 W Based on WO 9704097; AU 727480 B Previous Publ. AU 9667123,  
Based on WO 9704097; AU 727489 B Previous Publ. AU 9667685, Based on WO  
9707198; AU 2001028001 A Div ex AU 727489; AU 2001028002 A Div ex AU  
727480  
PRAI US 1995-514014 19950811; US 1995-504032 19950719; US 1997-833823  
19970410; WO 1996-US12897 19960808; AU 2001-28001 20010314; AU  
2001-28002 20010314  
AB WO 9704097 A UPAB: 20011001  
A novel isolated polynucleotide (I) comprises: (a) nucleotides (nt)  
146-544 of an 813 bp sequence given in the specification; (b) a sequence  
able to hybridise with (a) or varying from (a) only within the degeneracy  
of the genetic code; or (c) an allelic variant of (a). Also claimed are:  
(1) host cells transformed with (I); (2) isolated human CTLA-8 protein  
which has 163 amino acids (aa), its 11-163, 29-163 or 31-163 regions or  
any **fragments** of them with CTLA-8 activity; and (3)  
**antibodies** (Ab) which specifically react with CTLA-8 protein.  
USE - (I) encodes proteins with CTLA-8 activity. Treatment of mammals  
with CTLA-8 (or non-human analogues or IL-17) results in at least one of:  
(a) inhibition of angiogenesis, growth/proliferation of vascular  
endothelial cells, tumour cells and angiogenesis-dependent tissue growth;  
(b) proliferation of myeloid, erythroid or lymphoid cells (or their  
progeny); or (c) induction of interferon-gamma, IL-3 or GM-CSF prodn  
(claimed). Opt. CTLA-8 is expressed in vivo from a suitable vector.  
Typical applications of CTLA-8 are treatment of immune deficiency and  
disorders requiring modulation of T/B cell growth or proliferation, or of  
cytolytic natural killer cells, e.g. viral or microbial infection (e.g.  
HIV, hepatitis, malaria, candidiasis etc.); autoimmune disease (e.g.  
multiple sclerosis, rheumatoid **arthritis**, insulin-dependent  
diabetes etc.); to boost the immune response in cancer treatment; as  
antiinflammatories (e.g. in septic shock or Crohn's disease) and in  
haematopoietic disorders where growth/proliferation of erythroid, myeloid  
or megakaryocytic cells is needed. Ab can be used to determine CTLA-8,  
possibly also for treating some tumours or some of the above conditions.  
Dwg.0/7

**p40** production by anti-CD3 was abrogated by anti-CD154 antibody. **IL-12 p40** production was also increased by LPS stimulation. LPS-stimulated **IL-12** production was inhibited by anti-TNFalpha antibody, but not by T cell depletion and anti-CD154 antibody treatment. The TNFalpha inhibitor rolipram inhibited LPS-stimulated **IL-12** **p40** production by RA SC more strongly than spontaneous production. TNFalpha restored LPS-stimulated **IL-12** production that had been inhibited by rolipram. Conclusion: **IL-12** production in RA is regulated by 2 different pathways. One pathway is T cell dependent, predominantly through a CD40-CD154 interaction, while the other is T cell independent, mediated through TNFalpha. Inhibition of **IL-12** production by interference with CD40-CD154 interaction and TNFalpha production may be a potential therapeutic strategy for treating RA.

L19 ANSWER 2 OF 18 WPIDS (C) 2002 THOMSON DERWENT  
AN 2001-244560 [25] WPIDS  
DNC C2001-073385  
TI Composition comprising interleukin-12 **p40** and IL-B30 polypeptide or its segment, useful for ameliorating **rheumatoid arthritis**, osteoarthritis, atherosclerosis, multiple sclerosis, vasculitis and tumor.  
DC B04 D16  
IN DE WAAL MALEYTT, R; KASTELEIN, R A; LIRA, S A; NARULA, S K; OPPMANN, B; RENNICK, D M; WIEKOWSKI, M T  
PA (SCHE) SCHERING CORP  
CYC 92  
PI WO 2001018051 A2 20010315 (200125)\* EN 69p  
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ  
NL OA PT SD SE SL SZ TZ UG ZW  
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CR CZ DE DK DM DZ  
EE ES FI GB GD GE HR HU ID IL IN IS JP KG KR KZ LC LK LR LT LU LV  
MA MD MG MK MN MX MZ NO NZ PL PT RO RU SE SG SI SK SL TJ TM TR TT  
TZ UA UZ VN YU ZA  
AU 2000073608 A 20010410 (200137)  
EP 1210434 A2 20020605 (200238) EN  
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT  
RO SE SI  
ADT WO 2001018051 A2 WO 2000-US24686 20000908; AU 2000073608 A AU 2000-73608  
20000908; EP 1210434 A2 EP 2000-961688 20000908, WO 2000-US24686 20000908  
FDT AU 2000073608 A Based on WO 200118051; EP 1210434 A2 Based on WO 200118051  
PRAI US 1999-164616P 19991110; US 1999-393090 19990909  
AB WO 200118051 A UPAB: 20010508  
NOVELTY - A composition (I) comprising a substantially pure polypeptide comprising a number of distinct segments of at least 7 contiguous amino acids from interleukin (**IL**)-12 **p40** and/or IL-B30, and a substantially pure polypeptide comprising a segment of at least 11 contiguous amino acids from **IL-12 p40** and/or IL-B30.  
DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:  
(1) an isolated or recombinant nucleic acid (II) encoding (I);  
(2) a cell (III) comprising (II);  
(3) a nucleic acid (IV) which hybridizes under wash conditions of 30 minutes at 50 deg. C and less than 1M salt to the natural mature coding portion of primate **IL-12 p40** and IL-B30;  
(4) an antagonist (V) of **IL-12 p40** combined with a tumor necrosis factor-alpha (TNF alpha ) antagonist, an **IL-12 antagonist**, **IL-10**, or steroids;  
(5) a binding compound (VI) comprising an antigen binding site from

an **antibody**, which specifically binds to (I) and comprising a substantially pure polypeptide comprising **IL-12 p40** and IL-B30 polypeptide, or a polypeptide comprising **IL-12 p40** fused to IL-B30, but not to either **IL-12 p40** or IL-B30 polypeptide;

(6) a kit (VII) comprising:

(a) (I), and a compartment comprising the polypeptide, or instructions for use or disposal of reagents in the kit;

(b) (II), and a compartment comprising (II), a compartment further comprising a primate **IL-12 p40** or IL-B30, or instructions for use or disposal of reagents in the kit or (VI); and

(c) a compartment comprising (VI), or instructions for use or disposal of reagents in the kit;

(7) producing (M1) an antigen:**antibody** complex, involves contacting, under appropriate conditions, a primate **IL-12 p40/IL-B30** composition with (VI), allowing the complex to form;

(8) a composition (VIII) comprising (VI) which is sterile, or (VI) and a carrier such as an aqueous compound, including water, saline, and/or buffer;

(9) increasing (M2) the secretion of a primate IL-B30 involves expressing the polypeptide with **IL-12 p40** or increasing the secretion of a primate **IL-12 p40** involves expressing the **IL-12 p40** with IL-B30; and

(10) screening (M3) for a receptor which binds (I) involves contacting the complex to a cell expressing the receptor under conditions allowing the complex to bind to the receptor, forming a detectable interaction.

ACTIVITY - Antirheumatic; antiarthritic; osteopathic; antiarthritic; neuroprotective; antiarteriosclerotic; cerebroprotective; vasotropic; cytostatic; antitumor; immunosuppressive.

MECHANISM OF ACTION - Modulator of physiology or development of cell in host; inducer of memory T-cell proliferation (claimed); modulator of trafficking or activation of leukocyte.

No supporting data is given.

USE - (I) is useful for modulating physiology or development of a cell or tissue in a host organism by contacting the cell with (I) or (V), resulting in an increased or decreased production of Interferon-gamma (IFN gamma ), an enhanced Th1 response such as anti-tumor effect, adjuvant effect, anti-viral effect or antagonized allergic effect, and amelioration of an autoimmune condition or a chronic inflammatory condition. The contacting is in combination with IL-18, **IL-12**, radiation therapy or chemotherapy, an immune adjuvant or an anti-viral therapeutic. The **antagonist** is an **antibody** against **IL-12** receptor subunit beta 1. The **antagonist** or agonist of mammalian IL-B30 protein is useful for modulating the inflammatory response in an animal, by contacting cells in the animal with the agonist or **antagonist**, where the animal exhibits signs or symptoms of an acute phase inflammatory response in skin, lung, gastrointestinal, or liver tissue. The modulation is accelerating maturation of neutrophils into platelets and has an effect on immunoglobulin A and G (IgA and IgG) . The **antagonist** is an **antibody** which binds to the mammalian IL-B30 or blocks signaling mediated by mammalian IL-B30. The **antagonist** or agonist is administered in combination with an anti-inflammatory cytokine agonist or **antagonist**, an analgesic, an anti-inflammatory agent, or a steroid. IL-B30 or its agonist is useful inducing the proliferation of memory T-cells (all claimed).

Agonist or **antagonist** of IL-B30 protein is useful for modulating the trafficking or activation of a leukocyte in an animal experiencing science or symptoms of autoimmunity, an inflammatory

or heteroaryl; each of R.<sub>sub.2</sub>, R.<sub>sub.4</sub>, and R.<sub>sub.5</sub>, independently, is R.sup.c, halogen, nitro, nitroso, cyano, azide, isothionitro, SR.sup.c, or OR.sup.c; R.<sub>sub.3</sub> is R.sup.c, alkenyl, alkynyl, aryl, heteroaryl, cyclyl, heterocyclyl, OR.sup.c, OC(O)R.sup.c, SO<sub>2</sub>R.sup.c, S(O)R.sup.c, S(O)<sub>2</sub>NR.sup.c, NR.sup.cR.sup.d, SR.sup.c, NR.sup.cR.sup.d, NR.sup.cCOR.sup.d, NR.sup.cC(O)OR.sup.d, NR.sup.cC(O)NR.sup.cR.sup.d, NR.sup.cSO<sub>2</sub>R.sup.d, COR.sup.c, C(O)OR.sup.c, or C(O)NR.sup.cR.sup.d; n is 0, 1, 2, 3, 4, 5, 6, or 7; X is O, S, S(O), S(O)<sub>2</sub>, or NR.sup.c; Y is a covalent bond, CH<sub>2</sub>, C(O), C.dbd.N--R.sup.c, C.dbd.N--OR.sup.c, C.dbd.N--SR.sup.c, O, S, S(O), or S(O)<sub>2</sub>; Z is N; and W is O, S, S(O), S(O)<sub>2</sub>, NR.sup.c, or NC(O)R.sup.c; in which each of R.<sub>sup.a</sub> and R.<sub>sup.b</sub>, independently, is H, alkyl, aryl, heteroaryl; and each of R.<sub>sup.c</sub> and R.<sub>sup.d</sub>, independently, is H, alkyl, or alkylcarbonyl.

L19 ANSWER 5 OF 18 USPATFULL  
AN 2001:229210 USPATFULL  
TI Methods for enhancing oral tolerance and treating autoimmune disease using inhibitors of interleukin-12  
IN Strober, Warren, Bethesda, MD, United States  
Kelsall, Brian, Washington, DC, United States  
Marth, Thomas, Kensington, MD, United States  
PA Government of the United States of America, Department of Health and Human Services (U.S. corporation)  
PI US 2001051159 A1 20011213  
AI US 2000-732502 A1 20001207 (9)  
RLI Continuation of Ser. No. US 1999-284169, filed on 9 Apr 1999, ABANDONED  
A 371 of International Ser. No. WO 1996-US16007, filed on 11 Oct 1996,  
UNKNOWN  
DT Utility  
FS APPLICATION  
LREP mary l. miller THE CANDLER BUILDING, needle & rosenberg, p.c., 127  
peachtree street, n.e., atlanta, GA, 30303-1811  
CLMN Number of Claims: 20  
ECL Exemplary Claim: 1  
DRWN No Drawings  
LN.CNT 1252  
CAS INDEXING IS AVAILABLE FOR THIS PATENT.  
AB The present invention provides a method for enhancing oral tolerance to an antigen associated with an autoimmune disease in a subject having the autoimmune disease comprising orally administering to the subject an antigen associated with the autoimmune disease and administering an inhibitor of interleukin-12 in amounts sufficient to enhance oral tolerance. Also provided in the present invention is a method for treating or preventing an autoimmune disease in a subject comprising orally administering to the subject an antigen associated with the autoimmune disease and administering an inhibitor of interleukin-12 in amounts sufficient to treat or prevent the autoimmune disease, thereby treating or preventing the autoimmune disease.

L19 ANSWER 6 OF 18 USPATFULL  
AN 2001:221075 USPATFULL  
TI Retinoid antagonists and use thereof  
IN Bollag, Werner, Basel, Switzerland  
Klaus, Michael, Weil am Rhein, Germany, Federal Republic of  
Mohr, Peter, Basel, Switzerland  
Panina-Bordignon, Paola, Milan, Italy  
Rosenberger, Michael, Caldwell, NJ, United States  
Sinigaglia, Francesco, Milan, Italy  
PA Hoffman-La Roche Inc., Nutley, NJ, United States (U.S. corporation)  
PI US 6326397 B1 20011204

AI US 1999-307009 19990507 (9)  
RLI Continuation-in-part of Ser. No. US 1998-189189, filed on 10 Nov 1998

DT Utility  
FS GRANTED

EXNAM Primary Examiner: Killos, Paul J.  
LREP Johnston, George W., Parise, John P.  
CLMN Number of Claims: 16  
ECL Exemplary Claim: 1  
DRWN 7 Drawing Figure(s); 5 Drawing Page(s)

LN.CNT 1573

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention relates to novel retinoid antagonists of the formula I ##STR1##

wherein the dotted bond can be either hydrogenated or form a double bond; and, when the dotted bond forms a double bond, R.sup.1 is lower alkyl and R.sup.2 is hydrogen; and, when the dotted bond is hydrogenated, R.sup.1 and R.sup.2 taken together are methylene to form a cis-substituted cyclopropyl ring; R.sup.3 is hydroxy or lower alkoxy; R.sup.4 is alkyl or alkoxy; and R.sup.5 and R.sup.6 are, independently, a C.sub.4-12 alkyl or a 5-12 cycloalkyl substituent containing from 1-3 rings which are either unsubstituted or substituted with from 1-3 lower alkyl groups, with the carbon atom of R.sup.5 and R.sup.6 being linked to the remainder of the molecule to form a quaternary carbon atom pharmaceutically acceptable salts of carbocyclic acids of the formula I; as well as method for the treatment of osteoporosis and preneoplastic and neoplastic diseases, and a method for reducing or abolishing adverse events in subjects receiving retinoid agonist treatment by administering a retinoid antagonist.

L19 ANSWER 7 OF 18 USPATFULL

AN 2001:63494 USPATFULL

TI Antibodies against human IL-12

IN Gately, Maurice Kent, Parsippany, NJ, United States  
Presky, David Howard, Glen Ridge, NJ, United States

PA Hoffman-La Roche Inc., Nutley, NJ, United States (U.S. corporation)

PI US 6225117 B1 20010501

AI US 1999-232522 19990119 (9)

PRAI US 1998-72333P 19980123 (60)

DT Utility

FS Granted

EXNAM Primary Examiner: Chan, Christina Y.; Assistant Examiner: DiBrino, Marianne

LREP Johnston, George W., Rocha-Tramaloni, Patricia S., Silverman, Robert A.

CLMN Number of Claims: 23

ECL Exemplary Claim: 1

DRWN 7 Drawing Figure(s); 7 Drawing Page(s)

LN.CNT 1122

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention relates to novel p75 heterodimer specific anti-human IL-12 antibodies that are characterized by a higher potency and greater efficacy in neutralizing human IL-12 bioactivity than known heterodimer specific IL-12 monoclonal antibodies. The heterodimer specific antibodies recognize one or more epitopes of the human IL-12 p75 heterodimer, but do not bind to the p40 subunit alone. The heterodimer specific IL-12 antibodies neutralize rhesus monkey IL-12 bioactivity with a potency similar to their potency for neutralizing human IL-12 bioactivity making them useful IL-12 antagonists for in vivo studies in the rhesus monkey.

L19 ANSWER 8 OF 18 USPATFULL  
AN 2000:138395 USPATFULL  
TI Treatment of T-helper cell type 2-mediated immune disease by retinoid antagonists  
IN Bollag, Werner, Basel, Switzerland  
Klaus, Michael, Weil am Rhein, Germany, Federal Republic of  
Panina-Bordignon, Paola, Milan, Italy  
Sinigaglia, Francesco, Milan, Italy  
PA Hoffmann-La Roche Inc., Nutley, NJ, United States (U.S. corporation)  
PI US 6133309 20001017  
AI US 1998-189189 19981110 (9)  
PRAI EP 1997-119776 19971112  
DT Utility  
FS Granted  
EXNAM Primary Examiner: Travers, Russell  
LREP Johnston, George W., Epstein, William H., Parise, John P.  
CLMN Number of Claims: 37  
ECL Exemplary Claim: 1  
DRWN No Drawings  
LN.CNT 780  
CAS INDEXING IS AVAILABLE FOR THIS PATENT.  
AB Retinoids with retinoid receptor antagonistic activity, pharmaceutically acceptable salts and pharmaceutically acceptable hydrolyzable esters thereof, have been found efficacious in treating T-helper cell type 2 (Th2)-mediated immune diseases, such as immunoglobulin E (IgE)-mediated allergic diseases.

L19 ANSWER 9 OF 18 USPATFULL  
AN 2000:98551 USPATFULL  
TI Treatment of papillomavirus-associated lesions  
IN Stanley, Margaret Anne, Cambridge, United Kingdom  
Scarpini, Cinzia Giuseppina, Cambridge, United Kingdom  
PA Cambridge University Technical Services, Ltd., Cambridge, United Kingdom (non-U.S. corporation)  
PI US 6096869 20000801  
AI US 1996-621841 19960322 (8)  
PRAI GB 1995-5784 19950322  
DT Utility  
FS Granted  
EXNAM Primary Examiner: Park, Hankyel  
LREP Klarquist Sparkman Campbell Leigh & Whinston, LLP  
CLMN Number of Claims: 13  
ECL Exemplary Claim: 1  
DRWN No Drawings  
LN.CNT 1293  
CAS INDEXING IS AVAILABLE FOR THIS PATENT.  
AB Interleukin-12 (IL-12) or a functional analogue thereof, or a polynucleotide encoding IL-12 or encoding a functional analogue thereof, is used as a therapeutic material or adjuvant in treating papillomavirus-associated lesions e.g. warts due to HPV 6 and/or 11, e.g. condyloma acuminata. IL-12 or a vector encoding it for endogenous production can be used together with a vaccine such as a papillomavirus antigen, or a vector encoding a papillomavirus antigen.

L19 ANSWER 10 OF 18 USPATFULL  
AN 2000:87729 USPATFULL  
TI Method of converting a Th2-type allergic immune response into a Th1-type immune response  
IN DeKruyff, Rosemarie H., Stanford, CA, United States  
Umetsu, Dale T., Stanford, CA, United States

PA The Board of Trustees of the Leland Stanford Junior University, Palo Alto, CA, United States (U.S. corporation)  
PI US 6086898 20000711  
AI US 1999-339068 19990623 (9)  
PRAI US 1998-90390P 19980623 (60)  
DT Utility  
FS Granted  
EXNAM Primary Examiner: Chan, Christina Y.; Assistant Examiner: Ewoldt, Gerald R.  
LREP Bozicevic, Field & Francis, Sherwood, Pamela  
CLMN Number of Claims: 19  
ECL Exemplary Claim: 1  
DRWN 17 Drawing Figure(s); 10 Drawing Page(s)  
LN.CNT 1767

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Methods are provided for the treatment of allergic and other immune disorders associated with overproduction of Th2 type cytokines by antigen specific T cells. Immunotherapy with adjuvants, as provided in the present invention, greatly inhibits the development of airway hyperreactivity and airway inflammation. Such immunotherapy is shown to reverse ongoing airway disease, and convert allergic inflammatory responses into protective immune responses. Conditions of particular interest include allergic conditions associated with production of Th2 cytokines and/or IgE **antibodies**, asthma, allergic rhinitis, and anaphylactic reactions. The addition of adjuvant induces a Th1-type immune response and can redirect an established Th2-type response to a Th1-type response for the selected antigen. Preferably, antigen-specific IgE production is reduced without altering the intensity of the antigen-specific proliferative response. One particularly preferred adjuvant for use in accordance with the present invention is a Listeria adjuvant.

L19 ANSWER 11 OF 18 USPATFULL  
AN 2000:87707 USPATFULL  
TI Methods and compositions for the inhibition of interleukin-12 production  
IN Karp, Christopher L., Lutherville, MD, United States  
Trinchieri, Giorgio, Wynnewood, PA, United States  
Wysocka, Maria, Wynnewood, PA, United States  
Griffin, Diane E., Hunt Valley, MD, United States  
PA The Wistar Institute, Philadelphia, PA, United States (U.S. corporation)  
Johns Hopkins University, Baltimore, MD, United States (U.S. corporation)  
PI US 6086876 20000711  
AI US 1998-19862 19980206 (9)  
PRAI US 1997-37722P 19970207 (60)  
DT Utility  
FS Granted  
EXNAM Primary Examiner: Kemmerer, Elizabeth; Assistant Examiner: Romeo, David S.  
LREP Akin, Gump, Strauss, Hauer & Feld, L.L.P.  
CLMN Number of Claims: 12  
ECL Exemplary Claim: 1  
DRWN 13 Drawing Figure(s); 18 Drawing Page(s)  
LN.CNT 1487

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The invention includes compositions and methods for selective suppression of **IL-12** production in a cell. Methods of treating a human having a disease associated with dysregulated **IL-12** production are also provided.

L19 ANSWER 12 OF 18 USPATFULL  
AN 2000:50737 USPATFULL

formula ##STR1## wherein A is a single or double bond,  
B.sup.1 and B.sup.2 are each independently CH.dbd.CH or C.tbd.C,  
T is CH.sub.2 or CH.sub.2 CH.sub.2,  
X is --CH.sub.2 -- or >C.dbd.CH.sub.2,  
R.sup.1 is H, F or OH,  
R.sup.2 and R.sup.3 are each independently lower alkyl or CF.sub.3, or  
C(R.sup.2,R.sup.3) is C.sub.3-6 -cycloalkyl,  
are useful in the treatment or prevention of vitamin D dependent disorders and of **IL-12**-dependent autoimmune diseases, particularly psoriasis, basal cell carcinomas, disorders of keratinization and keratosis, leukemia, osteoporosis, hyperparathyroidism accompanying renal failure, multiple sclerosis, transplant rejection, graft vs. host disease, **rheumatoid arthritis**, insulin-dependent diabetes mellitus, inflammatory bowel disease, septic shock and allergic encephalomyelitis.

L19 ANSWER 14 OF 18 USPATFULL  
AN 1999:75759 USPATFULL  
TI Low affinity human **IL-12** beta2 receptor  
IN Gubler, Ulrich Andreas, Glen Ridge, NJ, United States  
Presky, David Howard, Glen Ridge, NJ, United States  
PA Hoffmann-La Roche Inc., Nutley, NJ, United States (U.S. corporation)  
PI US 5919903 19990706  
AI US 1997-914520 19970819 (8)  
RLI Division of Ser. No. US 1996-685118, filed on 23 Jul 1996  
PRAI US 1995-1701P 19950801 (60)  
DT Utility  
FS Granted  
EXNAM Primary Examiner: Draper, Garnette D.  
LREP Johnston, George W., Rocha-Tramaloni, Patricia S., Silverman, Robert A.  
CLMN Number of Claims: 2  
ECL Exemplary Claim: 1  
DRWN No Drawings  
LN.CNT 1531  
CAS INDEXING IS AVAILABLE FOR THIS PATENT.  
AB A recombinant human **IL-12** receptor complex produced on the surface of a non-human mammalian cell and free from other human proteins, the complex comprising the betal receptor protein complexed with a beta2 receptor protein, which complex is capable of binding to human **IL-12** with high affinity. A recombinant human **IL-12** beta2 receptor protein produced on the surface of a non-human mammalian cell, free from other human proteins, in its active form. In addition, a non-human mammalian cell having expressed on its surface the recombinant human **IL-12** beta2 receptor protein or the recombinant human **IL-12** receptor complex, which cell proliferates in the presence of human **IL-12**. A non-human mammalian cell having the human **IL-12** beta2 receptor protein or the complex expressed on its surface and which proliferates in response to human **IL-12** is useful for determining whether a given compound inhibits biological activity of human **IL-12** or is an **IL-12** agonist.

L19 ANSWER 15 OF 18 USPATFULL  
AN 1998:160106 USPATFULL

TI      **Antibodies** to receptors for human interleukin-12  
IN      Gubler, Ulrich Andreas, Glen Ridge, NJ, United States  
      Presky, David Howard, Glen Ridge, NJ, United States  
PA      Hoffmann-La Roche Inc., Nutley, NJ, United States (U.S. corporation)  
PI      US 5852176                          19981222  
AI      US 1997-915495                          19970820 (8)  
RLI     Division of Ser. No. US 1996-685118, filed on 23 Jul 1996  
PRAI    US 1995-1701P                          19950801 (60)  
DT      Utility  
FS      Granted  
EXNAM Primary Examiner: Draper, Garnette D.  
LREP Johnston, George W., Rocha-Tramaloni, Patricia S., Silverman, Robert A.  
CLMN Number of Claims: 1  
ECL Exemplary Claim: 1  
DRWN No Drawings  
LN.CNT 1381

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB      **Antibodies** to human **IL-12** beta 2 receptor  
protein or an **IL-12** receptor complex, the complex  
comprising the betal receptor protein complexed with a beta2 receptor  
protein, which complex is capable of binding to human **IL-**  
**12** with high affinity.

L19 ANSWER 16 OF 18 USPATFULL  
AN 1998:147252 USPATFULL  
TI DNA encoding receptors for the beta-2 chain of human **IL-**  
**12**  
IN Gubler, Ulrich Andreas, Glen Ridge, NJ, United States  
      Presky, David Howard, Glen Ridge, NJ, United States  
PA Hoffmann-La Roche Inc., Nutley, NJ, United States (U.S. corporation)  
PI US 5840530                          19981124  
AI US 1996-685118                          19960723 (8)  
PRAI US 1995-1701P                          19950801 (60)  
      US 1996-18674P                          19960530 (60)  
DT Utility  
FS Granted  
EXNAM Primary Examiner: Draper, Garnette D.  
LREP Johnston, George W., Rocha-Tramaloni, Patricia S., Silverman, Robert A.  
CLMN Number of Claims: 12  
ECL Exemplary Claim: 1  
DRWN No Drawings  
LN.CNT 1424

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB      A recombinant human **IL-12** beta2 receptor protein  
produced on the surface of a non-human mammalian cell, free from other  
human proteins, in its active form. In addition, a non-human mammalian  
cell having expressed on its surface the recombinant human **IL-**  
**12** beta2 receptor protein, which cell proliferates in the  
presence of human **IL-12**. A non-human mammalian cell  
having the human **IL-12** beta2 receptor protein on its  
surface and which proliferates in response to human **IL-**  
**12** is useful for determining whether a given compound inhibits  
biological activity of human **IL-12** or is an  
**IL-12** agonist.

L19 ANSWER 17 OF 18 USPATFULL  
AN 1998:135151 USPATFULL  
TI Human receptor for interleukin-12  
IN Chua, Anne On, Wayne, NJ, United States  
      Gubler, Ulrich Andreas, Glen Ridge, NJ, United States  
PA Hoffmann-La Roche Inc., Nutley, NJ, United States (U.S. corporation)  
PI US 5831007                          19981103

AI US 1995-419652 19950411 (8)  
RLI Division of Ser. No. US 1994-248532, filed on 31 May 1994, now patented,  
Pat. No. US 5536657 which is a continuation-in-part of Ser. No. US  
1993-94713, filed on 19 Jul 1993, now abandoned  
DT Utility  
FS Granted  
EXNAM Primary Examiner: Ulm, John  
LREP Johnston, George W., Epstein, William H., Bucholz, Briana C.  
CLMN Number of Claims: 10  
ECL Exemplary Claim: 1  
DRWN 35 Drawing Figure(s); 26 Drawing Page(s)  
LN.CNT 1937  
CAS INDEXING IS AVAILABLE FOR THIS PATENT.  
AB This invention relates to substantially pure Interleukin-12 receptor  
cDNAs and protein and uses therefore. The Interleukin-12 receptor is  
shown to be a member of the cytokine receptor superfamily and has a high  
homology to human gp130.

L19 ANSWER 18 OF 18 USPATFULL  
AN 96:63048 USPATFULL  
TI Recombinant DNA encoding human receptor for interleukin-12  
IN Chua, Anne O., Wayne, NJ, United States  
Gubler, Ulrich A., Glen Ridge, NJ, United States  
PA Hoffmann-La Roche Inc., Nutley, NJ, United States (U.S. corporation)  
PI US 5536657 19960716  
AI US 1994-248532 19940531 (8)  
RLI Continuation-in-part of Ser. No. US 1993-94713, filed on 19 Jul 1993,  
now abandoned  
DT Utility  
FS Granted  
EXNAM Primary Examiner: Ulm, John  
LREP Gould, George M., Johnston, George W., Kass, Alan P.  
CLMN Number of Claims: 10  
ECL Exemplary Claim: 1  
DRWN 34 Drawing Figure(s); 25 Drawing Page(s)  
LN.CNT 1755  
CAS INDEXING IS AVAILABLE FOR THIS PATENT.  
AB This invention relates to substantially pure Interleukin-12 receptor  
cDNAs and protein and uses therefore. The Interleukin-12 receptor is  
shown to be a member of the cytokine receptor superfamily and has a high  
homology to human gp130.

=> d his

(FILE 'HOME' ENTERED AT 11:40:44 ON 11 JUL 2002)

FILE 'BIOSIS, MEDLINE, AGRICOLA, EMBASE, CABAB, WPIDS, JAPIO, BIOTECHDS,  
LIFESCI, CAPLUS, USPATFULL, USPAT2' ENTERED AT 11:42:50 ON 11 JUL 2002  
E LEONARD JOHN P/AU  
L1 108 S E3-E5  
E LEONARD J P/AU  
L2 359 S E3-E4  
E GOLDMAN SAMUEL/AU  
L3 78 S E2-E9  
E GOLDMAN S/AU  
L4 1458 S E3  
E OHARA RICHARD/AU  
E OHARA R/AU  
L5 70 S E3  
L6 2059 S L1-L5  
L7 109 S L6 AND (IL-12 OR RA OR ARTHRITIS)

L8           43 S L7 AND (ANTIBOD? OR ANTAGONIST?)  
L9           20 DUP REM L8 (23 DUPLICATES REMOVED)  
L10          1 S L9 AND P40

FILE 'STNGUIDE' ENTERED AT 11:51:09 ON 11 JUL 2002  
L11          0 S L8 AND (FRAGMENT? OR EPITOP?)  
L12          2 S RA OR RHEUMATOID ARTHRITIS

FILE 'BIOSIS, MEDLINE, AGRICOLA, EMBASE, CABAB, WPIDS, JAPIO, BIOTECHDS,  
LIFESCI, CAPLUS, USPATFULL, USPAT2' ENTERED AT 11:55:50 ON 11 JUL 2002  
L13          3 S L9 AND (FRAGMENT? OR EPITOP?)  
L14          656173 S RA OR RHEUMATOID ARTHRITIS  
L15          1079 S L14 AND IL-12  
L16          724 S L15 AND (ANTIBOD? OR EPITOP? OR FRAGMENT? OR ANTAGONIST)  
L17          95 S L16 AND IL-12 (5A) ANTIBOD?  
L18          82 DUP REM L17 (13 DUPLICATES REMOVED)  
L19          18 S L18 AND P40

=> s l16 and IL-12 (5a) antagonist?  
L20          52 L16 AND IL-12 (5A) ANTAGONIST?

=> dup rem l20  
PROCESSING COMPLETED FOR L20  
L21          44 DUP REM L20 (8 DUPLICATES REMOVED)

=> d bib ab 1-44

L21 ANSWER 1 OF 44 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE  
1  
AN 2002:166945 BIOSIS  
DN PREV200200166945  
TI Use of **IL-12** and **IL-12**  
antagonists in the treatment of autoimmune diseases.  
AU Leonard, John (1); Goldman, Samuel; O'Hara, Richard, Jr.  
CS (1) Auburn, NH USA  
ASSIGNEE: Genetics Institute, Inc.  
PI US 6338848 January 15, 2002  
SO Official Gazette of the United States Patent and Trademark Office Patents,  
(Jan. 15, 2002) Vol. 1254, No. 3, pp. No Pagination.  
<http://www.uspto.gov/web/menu/patdata.html>. e-file.  
ISSN: 0098-1133.  
DT Patent  
LA English  
AB Method of treating autoimmune conditions are disclosed comprising  
administering to a mammalian subject **IL-12** or an  
**IL-12 antagonist**. In certain preferred  
embodiments the autoimmune condition is one which is promoted by an  
increase in levels of IFN-gamma or TNF-alpha. Suitable conditions for  
treatment include multiple sclerosis, systemic lupus erythematosus,  
**rheumatoid arthritis**, autoimmune pulmonary inflammation,  
Guillain-Barre syndrome, autoimmune thyroiditis, insulin dependent  
diabetes mellitus and autoimmune inflammatory eye disease.

L21 ANSWER 2 OF 44 USPATFULL  
AN 2002:165194 USPATFULL  
TI Nucleic acids, proteins, and **antibodies**  
IN Rosen, Craig A., Laytonsville, MD, UNITED STATES  
Ruben, Steven M., Olney, MD, UNITED STATES  
Barash, Steven C., Rockville, MD, UNITED STATES  
PI US 2002086823       A1   20020704  
AI US 2001-764889       A1   20010117 (9)  
PRAI US 2000-179065P     20000131 (60)

DT Utility  
FS APPLICATION  
LREP HUMAN GENOME SCIENCES INC, 9410 KEY WEST AVENUE, ROCKVILLE, MD, 20850  
CLMN Number of Claims: 24  
ECL Exemplary Claim: 1  
DRWN No Drawings  
LN.CNT 17471  
AB The present invention relates to novel respiratory system related polynucleotides and the polypeptides encoded by these polynucleotides herein collectively known as "respiratory system antigens," and the use of such respiratory system antigens for detecting disorders of the respiratory system, particularly the presence of cancer of respiratory system tissues and cancer metastases. More specifically, isolated respiratory system associated nucleic acid molecules are provided encoding novel respiratory system associated polypeptides. Novel respiratory system polypeptides and **antibodies** that bind to these polypeptides are provided. Also provided are vectors, host cells, and recombinant and synthetic methods for producing human respiratory system associated polynucleotides and/or polypeptides. The invention further relates to diagnostic and therapeutic methods useful for diagnosing, treating, preventing and/or prognosing disorders related to the respiratory system, including cancer of respiratory system tissues, and therapeutic methods for treating such disorders. The invention further relates to screening methods for identifying agonists and antagonists of polynucleotides and polypeptides of the invention. The present invention further relates to methods and/or compositions for inhibiting the production and function of the polypeptides of the present invention.

L21 ANSWER 3 OF 44 USPATFULL  
AN 2002:165193 USPATFULL  
TI Nucleic acids, proteins, and **antibodies**  
IN Rosen, Craig A., Laytonsville, MD, UNITED STATES  
Ruben, Steven M., Olney, MD, UNITED STATES  
Barash, Steven C., Rockville, MD, UNITED STATES  
PI US 2002086822 A1 20020704  
AI US 2001-764886 A1 20010117 (9)  
PRAI US 2000-179065P 20000131 (60)  
US 2000-180628P 20000204 (60)  
US 2000-214886P 20000628 (60)  
US 2000-217487P 20000711 (60)  
US 2000-225758P 20000814 (60)  
US 2000-220963P 20000726 (60)  
US 2000-217496P 20000711 (60)  
US 2000-225447P 20000814 (60)  
US 2000-218290P 20000714 (60)  
US 2000-225757P 20000814 (60)  
US 2000-226868P 20000822 (60)  
US 2000-216647P 20000707 (60)  
US 2000-225267P 20000814 (60)  
US 2000-216880P 20000707 (60)  
US 2000-225270P 20000814 (60)  
US 2000-251869P 20001208 (60)  
US 2000-235834P 20000927 (60)  
US 2000-234274P 20000921 (60)  
US 2000-234223P 20000921 (60)  
US 2000-228924P 20000830 (60)  
US 2000-224518P 20000814 (60)  
US 2000-236369P 20000929 (60)  
US 2000-224519P 20000814 (60)  
US 2000-220964P 20000726 (60)  
US 2000-241809P 20001020 (60)

US 2000-249299P	20001117 (60)
US 2000-236327P	20000929 (60)
US 2000-241785P	20001020 (60)
US 2000-244617P	20001101 (60)
US 2000-225268P	20000814 (60)
US 2000-236368P	20000929 (60)
US 2000-251856P	20001208 (60)
US 2000-251868P	20001208 (60)
US 2000-229344P	20000901 (60)
US 2000-234997P	20000925 (60)
US 2000-229343P	20000901 (60)
US 2000-229345P	20000901 (60)
US 2000-229287P	20000901 (60)
US 2000-229513P	20000905 (60)
US 2000-231413P	20000908 (60)
US 2000-229509P	20000905 (60)
US 2000-236367P	20000929 (60)
US 2000-237039P	20001002 (60)
US 2000-237038P	20001002 (60)
US 2000-236370P	20000929 (60)
US 2000-236802P	20001002 (60)
US 2000-237037P	20001002 (60)
US 2000-237040P	20001002 (60)
US 2000-240960P	20001020 (60)
US 2000-239935P	20001013 (60)

DT Utility

FS APPLICATION

LREP HUMAN GENOME SCIENCES INC, 9410 KEY WEST AVENUE, ROCKVILLE, MD, 20850

CLMN Number of Claims: 24

ECL Exemplary Claim: 1

DRWN No Drawings

LN.CNT 20931

AB The present invention relates to novel proteins. More specifically, isolated nucleic acid molecules are provided encoding novel polypeptides. Novel polypeptides and **antibodies** that bind to these polypeptides are provided. Also provided are vectors, host cells, and recombinant and synthetic methods for producing human polynucleotides and/or polypeptides, and **antibodies**. The invention further relates to diagnostic and therapeutic methods useful for diagnosing, treating, preventing and/or prognosing disorders related to these novel polypeptides. The invention further relates to screening methods for identifying agonists and antagonists of polynucleotides and polypeptides of the invention. The present invention further relates to methods and/or compositions for inhibiting or enhancing the production and function of the polypeptides of the present invention.

L21 ANSWER 4 OF 44 USPATFULL

AN 2002:165192 USPATFULL

TI Nucleic acids, proteins, and **antibodies**

IN Rosen, Craig A., Laytonsville, MD, UNITED STATES

Ruben, Steven M., Olney, MD, UNITED STATES

Barash, Steven C., Rockville, MD, UNITED STATES

PI US 2002086821 A1 20020704

AI US 2001-764881 A1 20010117 (9)

PRAI US 2000-179065P 20000131 (60)

DT Utility

FS APPLICATION

LREP HUMAN GENOME SCIENCES INC, 9410 KEY WEST AVENUE, ROCKVILLE, MD, 20850

CLMN Number of Claims: 24

ECL Exemplary Claim: 1

DRWN No Drawings

LN.CNT 27531

AB The present invention relates to novel respiratory system related polynucleotides and the polypeptides encoded by these polynucleotides herein collectively known as "respiratory system antigens," and the use of such respiratory system antigens for detecting disorders of the respiratory system, particularly the presence of cancer of respiratory system tissues and cancer metastases. More specifically, isolated respiratory system associated nucleic acid molecules are provided encoding novel respiratory system associated polypeptides. Novel respiratory system polypeptides and **antibodies** that bind to these polypeptides are provided. Also provided are vectors, host cells, and recombinant and synthetic methods for producing human respiratory system associated polynucleotides and/or polypeptides. The invention further relates to diagnostic and therapeutic methods useful for diagnosing, treating, preventing and/or prognosing disorders related to the respiratory system, including cancer of respiratory system tissues, and therapeutic methods for treating such disorders. The invention further relates to screening methods for identifying agonists and antagonists of polynucleotides and polypeptides of the invention. The present invention further relates to methods and/or compositions for inhibiting the production and function of the polypeptides of the present invention.

L21 ANSWER 5 OF 44 USPATFULL  
AN 2002:165191 USPATFULL  
TI Nucleic acids, proteins, and **antibodies**  
IN Rosen, Craig A., Laytonsville, MD, UNITED STATES  
Ruben, Steven M., Olney, MD, UNITED STATES  
Barash, Steven C., Rockville, MD, UNITED STATES  
PI US 2002086820 A1 20020704  
AI US 2001-764862 A1 20010117 (9)  
PRAI US 2000-179065P 20000131 (60)  
DT Utility  
FS APPLICATION  
LREP HUMAN GENOME SCIENCES INC, 9410 KEY WEST AVENUE, ROCKVILLE, MD, 20850  
CLMN Number of Claims: 24  
ECL Exemplary Claim: 1  
DRWN No Drawings  
LN.CNT 17727  
AB The present invention relates to novel respiratory system related polynucleotides and the polypeptides encoded by these polynucleotides herein collectively known as "respiratory system antigens," and the use of such respiratory system antigens for detecting disorders of the respiratory system, particularly the presence of cancer of respiratory system tissues and cancer metastases. More specifically, isolated respiratory system associated nucleic acid molecules are provided encoding novel respiratory system associated polypeptides. Novel respiratory system polypeptides and **antibodies** that bind to these polypeptides are provided. Also provided are vectors, host cells, and recombinant and synthetic methods for producing human respiratory system associated polynucleotides and/or polypeptides. The invention further relates to diagnostic and therapeutic methods useful for diagnosing, treating, preventing and/or prognosing disorders related to the respiratory system, including cancer of respiratory system tissues, and therapeutic methods for treating such disorders. The invention further relates to screening methods for identifying agonists and antagonists of polynucleotides and polypeptides of the invention. The present invention further relates to methods and/or compositions for inhibiting the production and function of the polypeptides of the present invention.

L21 ANSWER 6 OF 44 USPATFULL  
AN 2002:165182 USPATFULL

TI Nucleic acids, proteins, and **antibodies**  
IN Rosen, Craig A., Laytonsville, MD, UNITED STATES  
Ruben, Steven M., Olney, MD, UNITED STATES  
Barash, Steven C., Rockville, MD, UNITED STATES  
PI US 2002086811 Al 20020704  
AI US 2001-764861 Al 20010117 (9)  
PRAI US 2000-179065P 20000131 (60)  
US 2000-180628P 20000204 (60)  
US 2000-214886P 20000628 (60)  
US 2000-217487P 20000711 (60)  
US 2000-225758P 20000814 (60)  
US 2000-220963P 20000726 (60)  
US 2000-217496P 20000711 (60)  
US 2000-225447P 20000814 (60)  
US 2000-218290P 20000714 (60)  
US 2000-225757P 20000814 (60)  
US 2000-226868P 20000822 (60)  
US 2000-216647P 20000707 (60)  
US 2000-225267P 20000814 (60)  
US 2000-216880P 20000707 (60)  
US 2000-225270P 20000814 (60)  
US 2000-251869P 20001208 (60)  
US 2000-235834P 20000927 (60)  
US 2000-234274P 20000921 (60)  
US 2000-234223P 20000921 (60)  
US 2000-228924P 20000830 (60)  
US 2000-224518P 20000814 (60)  
US 2000-236369P 20000929 (60)  
US 2000-224519P 20000814 (60)  
US 2000-220964P 20000726 (60)  
US 2000-241809P 20001020 (60)  
US 2000-249299P 20001117 (60)  
US 2000-236327P 20000929 (60)  
US 2000-241785P 20001020 (60)  
US 2000-244617P 20001101 (60)  
US 2000-225268P 20000814 (60)  
US 2000-236368P 20000929 (60)  
US 2000-251856P 20001208 (60)  
US 2000-251868P 20001208 (60)  
US 2000-229344P 20000901 (60)  
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US 2000-229287P 20000901 (60)  
US 2000-229513P 20000905 (60)  
US 2000-231413P 20000908 (60)  
US 2000-229509P 20000905 (60)  
US 2000-236367P 20000929 (60)  
US 2000-237039P 20001002 (60)  
US 2000-237038P 20001002 (60)  
US 2000-236370P 20000929 (60)  
US 2000-236802P 20001002 (60)  
US 2000-237037P 20001002 (60)  
US 2000-237040P 20001002 (60)  
US 2000-240960P 20001020 (60)  
US 2000-239935P 20001013 (60)  
DT Utility  
FS APPLICATION  
LREP HUMAN GENOME SCIENCES INC, 9410 KEY WEST AVENUE, ROCKVILLE, MD, 20850  
CLMN Number of Claims: 24  
ECL Exemplary Claim: 1  
DRWN No Drawings

LN.CNT 22023

AB The present invention relates to novel proteins. More specifically, isolated nucleic acid molecules are provided encoding novel polypeptides. Novel polypeptides and **antibodies** that bind to these polypeptides are provided. Also provided are vectors, host cells, and recombinant and synthetic methods for producing human polynucleotides and/or polypeptides, and **antibodies**. The invention further relates to diagnostic and therapeutic methods useful for diagnosing, treating, preventing and/or prognosing disorders related to these novel polypeptides. The invention further relates to screening methods for identifying agonists and antagonists of polynucleotides and polypeptides of the invention. The present invention further relates to methods and/or compositions for inhibiting or enhancing the production and function of the polypeptides of the present invention.

L21 ANSWER 7 OF 44 USPATFULL  
AN 2002:164735 USPATFULL  
TI Nucleic acids, proteins, and **antibodies**  
IN Rosen, Craig A., Laytonsville, MD, UNITED STATES  
Ruben, Steven M., Olney, MD, UNITED STATES  
Barash, Steven C., Rockville, MD, UNITED STATES  
PI US 2002086353 A1 20020704  
AI US 2001-764856 A1 20010117 (9)  
PRAI US 2000-179065P 20000131 (60)  
US 2000-180628P 20000204 (60)  
US 2000-214886P 20000628 (60)  
US 2000-217487P 20000711 (60)  
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US 2000-229343P 20000901 (60)  
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US 2000-229513P 20000905 (60)

US 2000-231413P	20000908 (60)
US 2000-229509P	20000905 (60)
US 2000-236367P	20000929 (60)
US 2000-237039P	20001002 (60)
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US 2000-236802P	20001002 (60)
US 2000-237037P	20001002 (60)
US 2000-237040P	20001002 (60)
US 2000-240960P	20001020 (60)
US 2000-239935P	20001013 (60)

DT Utility

FS APPLICATION

LREP HUMAN GENOME SCIENCES INC, 9410 KEY WEST AVENUE, ROCKVILLE, MD, 20850

CLMN Number of Claims: 24

ECL Exemplary Claim: 1

DRWN No Drawings

LN.CNT 23314

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention relates to novel proteins. More specifically, isolated nucleic acid molecules are provided encoding novel polypeptides. Novel polypeptides and **antibodies** that bind to these polypeptides are provided. Also provided are vectors, host cells, and recombinant and synthetic methods for producing human polynucleotides and/or polypeptides, and **antibodies**. The invention further relates to diagnostic and therapeutic methods useful for diagnosing, treating, preventing and/or prognosing disorders related to these novel polypeptides. The invention further relates to screening methods for identifying agonists and antagonists of polynucleotides and polypeptides of the invention. The present invention further relates to methods and/or compositions for inhibiting or enhancing the production and function of the polypeptides of the present invention.

L21 ANSWER 8 OF 44 USPATFULL

AN 2002:164712 USPATFULL

TI Nucleic acids, proteins, and **antibodies**

IN Rosen, Craig A., Laytonsville, MD, UNITED STATES

Ruben, Steven M., Olney, MD, UNITED STATES

Barash, Steven C., Rockville, MD, UNITED STATES

PI US 2002086330 A1 20020704

AI US 2001-764893 A1 20010117 (9)

PRAI US 2000-179065P 20000131 (60)

US 2000-180628P 20000204 (60)

US 2000-214886P 20000628 (60)

US 2000-217487P 20000711 (60)

US 2000-225758P 20000814 (60)

US 2000-220963P 20000726 (60)

US 2000-217496P 20000711 (60)

US 2000-225447P 20000814 (60)

US 2000-218290P 20000714 (60)

US 2000-225757P 20000814 (60)

US 2000-226868P 20000822 (60)

US 2000-216647P 20000707 (60)

US 2000-225267P 20000814 (60)

US 2000-216880P 20000707 (60)

US 2000-225270P 20000814 (60)

US 2000-251869P 20001208 (60)

US 2000-235834P 20000927 (60)

US 2000-234274P 20000921 (60)

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US 2000-228924P 20000830 (60)

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US 2000-236369P	20000929 (60)
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US 2000-220964P	20000726 (60)
US 2000-241809P	20001020 (60)
US 2000-249299P	20001117 (60)
US 2000-236327P	20000929 (60)
US 2000-241785P	20001020 (60)
US 2000-244617P	20001101 (60)
US 2000-225268P	20000814 (60)
US 2000-236368P	20000929 (60)
US 2000-251856P	20001208 (60)
US 2000-251868P	20001208 (60)
US 2000-229344P	20000901 (60)
US 2000-234997P	20000925 (60)
US 2000-229343P	20000901 (60)
US 2000-229345P	20000901 (60)
US 2000-229287P	20000901 (60)
US 2000-229513P	20000905 (60)
US 2000-231413P	20000908 (60)
US 2000-229509P	20000905 (60)
US 2000-236367P	20000929 (60)
US 2000-237039P	20001002 (60)
US 2000-237038P	20001002 (60)
US 2000-236370P	20000929 (60)
US 2000-236802P	20001002 (60)
US 2000-237037P	20001002 (60)
US 2000-237040P	20001002 (60)
US 2000-240960P	20001020 (60)
US 2000-239935P	20001013 (60)

DT Utility

FS APPLICATION

LREP HUMAN GENOME SCIENCES INC, 9410 KEY WEST AVENUE, ROCKVILLE, MD, 20850

CLMN Number of Claims: 24

ECL Exemplary Claim: 1

DRWN No Drawings

LN.CNT 25862

AB The present invention relates to novel proteins. More specifically, isolated nucleic acid molecules are provided encoding novel polypeptides. Novel polypeptides and **antibodies** that bind to these polypeptides are provided. Also provided are vectors, host cells, and recombinant and synthetic methods for producing human polynucleotides and/or polypeptides, and **antibodies**. The invention further relates to diagnostic and therapeutic methods useful for diagnosing, treating, preventing and/or prognosing disorders related to these novel polypeptides. The invention further relates to screening methods for identifying agonists and antagonists of polynucleotides and polypeptides of the invention. The present invention further relates to methods and/or compositions for inhibiting or enhancing the production and function of the polypeptides of the present invention.

The present invention relates to novel proteins. More specifically, isolated nucleic acid molecules are provided encoding novel polypeptides. Novel polypeptides and **antibodies** that bind to these polypeptides are provided. Also provided are vectors, host cells, and recombinant and synthetic methods for producing human polynucleotides and/or polypeptides, and **antibodies**. The invention further relates to diagnostic and therapeutic methods useful for diagnosing, treating, preventing and/or prognosing disorders related to these novel polypeptides. The invention further relates to screening methods for identifying agonists and antagonists of polynucleotides and polypeptides of the invention. The present invention further relates to methods and/or compositions for inhibiting or enhancing the production

and function of the polypeptides of the present invention.

L21 ANSWER 9 OF 44 USPATFULL  
AN 2002:157060 USPATFULL  
TI Nucleic acids, proteins and **antibodies**  
IN Rosen, Craig A., Laytonsville, MD, UNITED STATES  
Ruben, Steven M., Olney, MD, UNITED STATES  
PI US 2002081659 A1 20020627  
AI US 2001-925297 A1 20010810 (9)  
RLI Continuation-in-part of Ser. No. WO 2000-US5989, filed on 8 Mar 2000,  
UNKNOWN  
PRAI US 1999-124270P 19990312 (60)  
DT Utility  
FS APPLICATION  
LREP HUMAN GENOME SCIENCES INC, 9410 KEY WEST AVENUE, ROCKVILLE, MD, 20850  
CLMN Number of Claims: 23  
ECL Exemplary Claim: 1  
DRWN No Drawings  
LN.CNT 20326

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention relates to novel pancreatic related polynucleotides, the polypeptides encoded by these polynucleotides herein collectively referred to as "pancreatic antigens," and **antibodies** that immunospecifically bind these polypeptides, and the use of such pancreatic polynucleotides, antigens, and **antibodies** for detecting, treating, preventing and/or prognosing disorders of the pancreas, including, but not limited to, the presence of pancreatic cancer and pancreatic cancer metastases. More specifically, isolated pancreatic nucleic acid molecules are provided encoding novel pancreatic polypeptides. Novel pancreatic polypeptides and **antibodies** that bind to these polypeptides are provided. Also provided are vectors, host cells, and recombinant and synthetic methods for producing human pancreatic polynucleotides, polypeptides, and/or **antibodies**. The invention further relates to diagnostic and therapeutic methods useful for diagnosing, treating, preventing and/or prognosing disorders related to the pancreas, including pancreatic cancer, and therapeutic methods for treating such disorders. The invention further relates to screening methods for identifying agonists and antagonists of polynucleotides and polypeptides of the invention. The invention further relates to methods and/or compositions for inhibiting or promoting the production and/or function of the polypeptides of the invention.

L21 ANSWER 10 OF 44 USPATFULL  
AN 2002:149114 USPATFULL  
TI Nucleic acids, proteins, and **antibodies**  
IN Rosen, Craig A., Laytonsville, MD, UNITED STATES  
Ruben, Steven M., Olney, MD, UNITED STATES  
Barash, Steven C., Rockville, MD, UNITED STATES  
PI US 2002077270 A1 20020620  
AI US 2001-764848 A1 20010117 (9)  
PRAI US 2000-179065P 20000131 (60)  
US 2000-180628P 20000204 (60)  
US 2000-214886P 20000628 (60)  
US 2000-217487P 20000711 (60)  
US 2000-225758P 20000814 (60)  
US 2000-220963P 20000726 (60)  
US 2000-217496P 20000711 (60)  
US 2000-225447P 20000814 (60)  
US 2000-218290P 20000714 (60)  
US 2000-225757P 20000814 (60)  
US 2000-226868P 20000822 (60)

US 2000-216647P	20000707 (60)
US 2000-225267P	20000814 (60)
US 2000-216880P	20000707 (60)
US 2000-225270P	20000814 (60)
US 2000-251869P	20001208 (60)
US 2000-235834P	20000927 (60)
US 2000-234274P	20000921 (60)
US 2000-234223P	20000921 (60)
US 2000-228924P	20000830 (60)
US 2000-224518P	20000814 (60)
US 2000-236369P	20000929 (60)
US 2000-224519P	20000814 (60)
US 2000-220964P	20000726 (60)
US 2000-241809P	20001020 (60)
US 2000-249299P	20001117 (60)
US 2000-236327P	20000929 (60)
US 2000-241785P	20001020 (60)
US 2000-244617P	20001101 (60)
US 2000-225268P	20000814 (60)
US 2000-236368P	20000929 (60)
US 2000-251856P	20001208 (60)
US 2000-251868P	20001208 (60)
US 2000-229344P	20000901 (60)
US 2000-234997P	20000925 (60)
US 2000-229343P	20000901 (60)
US 2000-229345P	20000901 (60)
US 2000-229287P	20000901 (60)
US 2000-229513P	20000905 (60)
US 2000-231413P	20000908 (60)
US 2000-229509P	20000905 (60)
US 2000-236367P	20000929 (60)
US 2000-237039P	20001002 (60)
US 2000-237038P	20001002 (60)
US 2000-236370P	20000929 (60)
US 2000-236802P	20001002 (60)
US 2000-237037P	20001002 (60)
US 2000-237040P	20001002 (60)
US 2000-240960P	20001020 (60)
US 2000-239935P	20001013 (60)

DT Utility

FS APPLICATION

LREP HUMAN GENOME SCIENCES INC, 9410 KEY WEST AVENUE, ROCKVILLE, MD, 20850

CLMN Number of Claims: 24

ECL Exemplary Claim: 1

DRWN No Drawings

LN.CNT 20057

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention relates to novel proteins. More specifically, isolated nucleic acid molecules are provided encoding novel polypeptides. Novel polypeptides and **antibodies** that bind to these polypeptides are provided. Also provided are vectors, host cells, and recombinant and synthetic methods for producing human polynucleotides and/or polypeptides, and **antibodies**. The invention further relates to diagnostic and therapeutic methods useful for diagnosing, treating, preventing and/or prognosing disorders related to these novel polypeptides. The invention further relates to screening methods for identifying agonists and antagonists of polynucleotides and polypeptides of the invention. The present invention further relates to methods and/or compositions for inhibiting or enhancing the production and function of the polypeptides of the present invention.

AN 2002:148255 USPATFULL  
TI Membrane-bound cytokine compositions and methods of modulating an immune response using same  
IN Hoo, William Soo, Carlsbad, CA, UNITED STATES  
PI US 2002076392 A1 20020620  
AI US 2001-847185 A1 20010501 (9)  
RLI Continuation of Ser. No. US 1998-201931, filed on 1 Dec 1998, PENDING  
Continuation of Ser. No. US 1997-902516, filed on 29 Jul 1997, GRANTED,  
Pat. No. US 5891432  
DT Utility  
FS APPLICATION  
LREP CAMPBELL & FLORES LLP, 4370 LA JOLLA VILLAGE DRIVE, 7TH FLOOR, SAN DIEGO, CA, 92122  
CLMN Number of Claims: 46  
ECL Exemplary Claim: 1  
DRWN 4 Drawing Page(s)  
LN.CNT 1978

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention provides a cellular vaccine having a membrane-bound fusion protein that includes a non-**antibody** immunomodulatory molecule operatively fused to a heterologous membrane attachment domain. Non-**antibody** immunomodulatory molecules useful in the invention include immunostimulatory and immunosuppressive molecules such as cytokines. In one embodiment, the invention provides a cellular vaccine having a membrane-bound fusion protein that includes a non-**antibody** immunomodulatory molecule operatively fused to a heterologous membrane attachment domain and, additionally, a disease-associated antigen or immunogenic **epitope** thereof. Further provided by the invention are methods of modulating an immune response against a disease-associated antigen by administering to an individual a cellular vaccine having a membrane-bound fusion protein that includes a non-**antibody** immunomodulatory molecule operatively fused to a heterologous membrane attachment domain.

L21 ANSWER 12 OF 44 USPATFULL  
AN 2002:133211 USPATFULL  
TI Cytokine antagonists  
IN Debets, Johannes Eduard Maria Antonius, Rhoon, NETHERLANDS  
Abrams, John S., Los Altos, CA, UNITED STATES  
Kastelein, Robert A., Redwood City, CA, UNITED STATES  
O'Garra, Anne, Palo Alto, CA, UNITED STATES  
PI US 2002068060 A1 20020606  
AI US 2001-834295 A1 20010412 (9)  
PRAI US 2000-196754P 20000412 (60)  
DT Utility  
FS APPLICATION  
LREP SCHERING-PLOUGH CORPORATION, PATENT DEPARTMENT (K-6-1, 1990), 2000 GALLOPING HILL ROAD, KENILWORTH, NJ, 07033-0530  
CLMN Number of Claims: 21  
ECL Exemplary Claim: 1  
DRWN No Drawings  
LN.CNT 862

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Antagonists of a cytokine signaling system have been found which exhibit favorable properties. In particular, **antibody** antagonists raised against the receptor are effective in blocking various signaling processes.

L21 ANSWER 13 OF 44 USPATFULL  
AN 2002:119538 USPATFULL  
TI Nucleic acids, proteins, and **antibodies**  
IN Rosen, Craig A., Laytonsville, MD, UNITED STATES

Ruben, Steven M., Olney, MD, UNITED STATES  
Barash, Steven C., Rockville, MD, UNITED STATES  
PI US 2002061521 A1 20020523  
AI US 2001-764869 A1 20010117 (9)  
PRAI US 2000-179065P 20000131 (60)  
DT Utility  
FS APPLICATION  
LREP HUMAN GENOME SCIENCES INC, 9410 KEY WEST AVENUE, ROCKVILLE, MD, 20850  
CLMN Number of Claims: 24  
ECL Exemplary Claim: 1  
DRWN No Drawings  
LN.CNT 27967

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention relates to novel cardiovascular system related polynucleotides and the polypeptides encoded by these polynucleotides herein collectively known as "cardiovascular system antigens," and the use of such cardiovascular system antigens for detecting disorders of the cardiovascular system, particularly the presence of cancer of cardiovascular system tissues and cancer metastases. More specifically, isolated cardiovascular system associated nucleic acid molecules are provided encoding novel cardiovascular system associated polypeptides. Novel cardiovascular system polypeptides and **antibodies** that bind to these polypeptides are provided. Also provided are vectors, host cells, and recombinant and synthetic methods for producing human cardiovascular system associated polynucleotides and/or polypeptides. The invention further relates to diagnostic and therapeutic methods useful for diagnosing, treating, preventing and/or prognosing disorders related to the cardiovascular system, including cancer of cardiovascular system tissues, and therapeutic methods for treating such disorders. The invention further relates to screening methods for identifying agonists and antagonists of polynucleotides and polypeptides of the invention. The present invention further relates to methods and/or compositions for inhibiting the production and function of the polypeptides of the present invention.

L21 ANSWER 14 OF 44 USPATFULL  
AN 2002:106416 USPATFULL  
TI Nucleic acids, proteins and **antibodies**  
IN Rosen, Craig A., Laytonsville, MD, UNITED STATES  
Ruben, Steven M., Olney, MD, UNITED STATES  
PI US 2002055627 A1 20020509  
AI US 2001-925299 A1 20010810 (9)  
RLI Continuation of Ser. No. WO 2000-US5883, filed on 8 Mar 2000, UNKNOWN  
PRAI US 1999-124270P 19990312 (60)  
DT Utility  
FS APPLICATION  
LREP HUMAN GENOME SCIENCES INC, 9410 KEY WEST AVENUE, ROCKVILLE, MD, 20850  
CLMN Number of Claims: 23  
ECL Exemplary Claim: 1  
DRWN No Drawings  
LN.CNT 20658

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention relates to novel colorectal cancer related polynucleotides, the polypeptides encoded by these polynucleotides herein collectively referred to as "colorectal cancer antigens," and **antibodies** that immunospecifically bind these polypeptides, and the use of such colorectal cancer polynucleotides, antigens, and **antibodies** for detecting, treating, preventing and/or prognosing disorders of the colon and/or rectum, including, but not limited to, the presence of colorectal cancer and colorectal cancer metastases. More specifically, isolated colorectal cancer nucleic acid molecules are provided encoding novel colorectal cancer polypeptides. Novel colorectal

cancer polypeptides and **antibodies** that bind to these polypeptides are provided. Also provided are vectors, host cells, and recombinant and synthetic methods for producing human colorectal cancer polynucleotides, polypeptides, and/or **antibodies**. The invention further relates to diagnostic and therapeutic methods useful for diagnosing, treating, preventing and/or prognosing disorders related to the colon and/or rectum, including colorectal cancer, and therapeutic methods for treating such disorders. The invention further relates to screening methods for identifying agonists and antagonists of polynucleotides and polypeptides of the invention. The invention further relates to methods and/or compositions for inhibiting or promoting the production and/or function of the polypeptides of the invention.

L21 ANSWER 15 OF 44 USPATFULL  
AN 2002:99407 USPATFULL  
TI Nucleic acids, proteins and **antibodies**  
IN Rosen, Craig A., Laytonsville, MD, UNITED STATES  
Ruben, Steven M., Olney, MD, UNITED STATES  
PI US 2002052308 A1 20020502  
AI US 2001-925301 A1 20010810 (9)  
RLI Continuation of Ser. No. WO 2000-US5882, filed on 8 Mar 2000, UNKNOWN  
PRAI US 1999-124270P 19990312 (60)  
DT Utility  
FS APPLICATION  
LREP HUMAN GENOME SCIENCES INC, 9410 KEY WEST AVENUE, ROCKVILLE, MD, 20850  
CLMN Number of Claims: 23  
ECL Exemplary Claim: 1  
DRWN No Drawings  
LN.CNT 30577  
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB This invention relates to newly identified tissue specific cancer associated polynucleotides and the polypeptides encoded by these polynucleotides herein collectively known as "cancer antigens," and to the complete gene sequences associated therewith and to the expression products thereof, as well as the use of such tissue specific cancer antigens for detection, prevention and treatment of tissue specific disorders, particularly the presence of cancer. This invention relates to the cancer antigens as well as vectors, host cells, **antibodies** directed to cancer antigens and recombinant and synthetic methods for producing the same. Also provided are diagnostic methods for diagnosing and treating, preventing and/or prognosing tissue specific disorders, including cancer, and therapeutic methods for treating such disorders. The invention further relates to screening methods for identifying agonists and antagonists of cancer antigens of the invention. The present invention further relates to methods and/or compositions for inhibiting the production and/or function of the polypeptides of the present invention.

L21 ANSWER 16 OF 44 USPATFULL  
AN 2002:85190 USPATFULL  
TI Nucleic acids, proteins, and **antibodies**  
IN Rosen, Craig A., Laytonsville, MD, UNITED STATES  
Rubin, Steven M., Olney, MD, UNITED STATES  
Barash, Steven C., Rockville, MD, UNITED STATES  
PI US 2002045230 A1 20020418  
AI US 2001-908711 A1 20010720 (9)  
RLI Continuation-in-part of Ser. No. WO 2001-US1360, filed on 17 Jan 2001, UNKNOWN Continuation-in-part of Ser. No. US 2001-764867, filed on 17 Jan 2001, UNKNOWN Continuation-in-part of Ser. No. WO 2001-US1344, filed on 17 Jan 2001, UNKNOWN Continuation-in-part of Ser. No. US 2001-764892, filed on 17 Jan 2001, UNKNOWN Continuation-in-part of Ser. No. WO 2001-US1345, filed on 17 Jan 2001, UNKNOWN Continuation-in-part of Ser.

No. US 2001-764888, filed on 17 Jan 2001, UNKNOWN Continuation-in-part of Ser. No. WO 2001-US1329, filed on 17 Jan 2001, UNKNOWN Continuation-in-part of Ser. No. US 2001-764905, filed on 17 Jan 2001, UNKNOWN Continuation-in-part of Ser. No. US 2001-764891, filed on 17 Jan 2001, UNKNOWN Continuation-in-part of Ser. No. WO 2001-US1339, filed on 17 Jan 2001, UNKNOWN Continuation-in-part of Ser. No. US 2001-764869, filed on 17 Jan 2001, UNKNOWN Continuation-in-part of Ser. No. WO 2001-US1340, filed on 17 Jan 2001, UNKNOWN Continuation-in-part of Ser. No. US 2001-764874, filed on 17 Jan 2001, UNKNOWN Continuation-in-part of Ser. No. WO 2001-US1334, filed on 17 Jan 2001, UNKNOWN Continuation-in-part of Ser. No. US 2001-764898, filed on 17 Jan 2001, UNKNOWN Continuation-in-part of Ser. No. WO 2001-US1320, filed on 17 Jan 2001, UNKNOWN Continuation-in-part of Ser. No. US 2001-764853, filed on 17 Jan 2001, UNKNOWN Continuation-in-part of Ser. No. US 2001-764902, filed on 17 Jan 2001, UNKNOWN Continuation-in-part of Ser. No. WO 2001-US1239, filed on 17 Jan 2001, UNKNOWN Continuation-in-part of Ser. No. US 2001-764870, filed on 17 Jan 2001, UNKNOWN Continuation-in-part of Ser. No. WO 2001-US1348, filed on 17 Jan 2001, UNKNOWN Continuation-in-part of Ser. No. US 2001-764882, filed on 17 Jan 2001, UNKNOWN Continuation-in-part of Ser. No. WO 2001-US1347, filed on 17 Jan 2001, UNKNOWN Continuation-in-part of Ser. No. US 2001-764896, filed on 17 Jan 2001, UNKNOWN Continuation-in-part of Ser. No. WO 2001-US1307, filed on 17 Jan 2001, UNKNOWN Continuation-in-part of Ser. No. US 2001-764864, filed on 17 Jan 2001, UNKNOWN Continuation-in-part of Ser. No. WO 2001-US1341, filed on 17 Jan 2001, UNKNOWN Continuation-in-part of Ser. No. US 2001-764856, filed on 17 Jan 2001, UNKNOWN Continuation-in-part of Ser. No. WO 2001-US1336, filed on 17 Jan 2001, UNKNOWN Continuation-in-part of Ser. No. US 2001-764868, filed on 17 Jan 2001, UNKNOWN Continuation-in-part of Ser. No. WO 2001-US1312, filed on 17 Jan 2001, UNKNOWN

PRAI	US 2000-179065P	20000131 (60)
	US 2000-180628P	20000204 (60)
	US 2000-251868P	20001208 (60)
	US 2000-232398P	20000914 (60)
	US 2000-249300P	20001117 (60)
	US 2000-251990P	20001208 (60)
	US 2000-250160P	20001201 (60)
	US 2000-209467P	20000607 (60)
	US 2000-179065P	20000131 (60)
	US 2000-180628P	20000204 (60)
	US 2000-214886P	20000628 (60)
	US 2000-217487P	20000711 (60)
	US 2000-225758P	20000814 (60)
	US 2000-220963P	20000726 (60)
	US 2000-217496P	20000711 (60)
	US 2000-225447P	20000814 (60)
	US 2000-218290P	20000714 (60)
	US 2000-225757P	20000814 (60)
	US 2000-226868P	20000822 (60)
	US 2000-216647P	20000707 (60)
	US 2000-225267P	20000814 (60)
	US 2000-216880P	20000707 (60)
	US 2000-225270P	20000814 (60)
	US 2000-251869P	20001208 (60)
	US 2000-235834P	20000927 (60)
	US 2000-234274P	20000921 (60)
	US 2000-234223P	20000921 (60)
	US 2000-228924P	20000830 (60)
	US 2000-224518P	20000814 (60)
	US 2000-236369P	20000929 (60)
	US 2000-224519P	20000814 (60)
	US 2000-220964P	20000726 (60)

US 2000-241809P	20001020 (60)
US 2000-249299P	20001117 (60)
US 2000-236327P	20000929 (60)
US 2000-241785P	20001020 (60)
US 2000-244617P	20001101 (60)
US 2000-225268P	20000814 (60)
US 2000-236368P	20000929 (60)
US 2000-251856P	20001208 (60)
US 2000-251868P	20001208 (60)
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US 2000-229343P	20000901 (60)
US 2000-229345P	20000901 (60)
US 2000-229287P	20000901 (60)
US 2000-229513P	20000905 (60)
US 2000-231413P	20000908 (60)
US 2000-229509P	20000905 (60)
US 2000-236367P	20000929 (60)
US 2000-237039P	20001002 (60)
US 2000-237038P	20001002 (60)
US 2000-236370P	20000929 (60)
US 2000-236802P	20001002 (60)
US 2000-237037P	20001002 (60)
US 2000-237040P	20001002 (60)
US 2000-240960P	20001020 (60)
US 2000-239935P	20001013 (60)
US 2000-239937P	20001013 (60)
US 2000-241787P	20001020 (60)
US 2000-246474P	20001108 (60)
US 2000-246532P	20001108 (60)
US 2000-249216P	20001117 (60)
US 2000-249210P	20001117 (60)
US 2000-226681P	20000822 (60)
US 2000-225759P	20000814 (60)
US 2000-225213P	20000814 (60)
US 2000-227182P	20000822 (60)
US 2000-225214P	20000814 (60)
US 2000-235836P	20000927 (60)
US 2000-230438P	20000906 (60)
US 2000-215135P	20000630 (60)
US 2000-225266P	20000814 (60)
US 2000-249218P	20001117 (60)
US 2000-249208P	20001117 (60)
US 2000-249213P	20001117 (60)
US 2000-249212P	20001117 (60)
US 2000-249207P	20001117 (60)
US 2000-249245P	20001117 (60)
US 2000-249244P	20001117 (60)
US 2000-249217P	20001117 (60)
US 2000-249211P	20001117 (60)
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US 2000-249264P	20001117 (60)
US 2000-249214P	20001117 (60)
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US 2000-232400P	20000914 (60)
US 2000-231242P	20000908 (60)
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US 2000-232080P	20000908 (60)
US 2000-231414P	20000908 (60)
US 2000-231244P	20000908 (60)
US 2000-233064P	20000914 (60)
US 2000-233063P	20000914 (60)

US 2000-232397P	20000914 (60)
US 2000-232399P	20000914 (60)
US 2000-232401P	20000914 (60)
US 2000-241808P	20001020 (60)
US 2000-241826P	20001020 (60)
US 2000-241786P	20001020 (60)
US 2000-241221P	20001020 (60)
US 2000-246475P	20001108 (60)
US 2000-231243P	20000908 (60)
US 2000-233065P	20000914 (60)
US 2000-232398P	20000914 (60)
US 2000-234998P	20000925 (60)
US 2000-246477P	20001108 (60)
US 2000-246528P	20001108 (60)
US 2000-246525P	20001108 (60)
US 2000-246476P	20001108 (60)
US 2000-246526P	20001108 (60)
US 2000-249209P	20001117 (60)
US 2000-246527P	20001108 (60)
US 2000-246523P	20001108 (60)
US 2000-246524P	20001108 (60)
US 2000-246478P	20001108 (60)
US 2000-246609P	20001108 (60)
US 2000-246613P	20001108 (60)
US 2000-249300P	20001117 (60)
US 2000-249265P	20001117 (60)
US 2000-246610P	20001108 (60)
US 2000-246611P	20001108 (60)
US 2000-230437P	20000906 (60)
US 2000-251990P	20001208 (60)
US 2000-251988P	20001205 (60)
US 2000-251030P	20001205 (60)
US 2000-251479P	20001206 (60)
US 2000-256719P	20001205 (60)
US 2000-250160P	20001201 (60)
US 2000-251989P	20001208 (60)
US 2000-250391P	20001201 (60)
US 2000-254097P	20001211 (60)
US 2000-231968P	20000912 (60)
US 2000-226279P	20000818 (60)
US 2000-186350P	20000302 (60)
US 2000-184664P	20000224 (60)
US 2000-189874P	20000316 (60)
US 2000-198123P	20000418 (60)
US 2000-227009P	20000823 (60)
US 2000-235484P	20000926 (60)
US 2000-190076P	20000317 (60)
US 2000-209467P	20000607 (60)
US 2000-205515P	20000519 (60)
US 2001-259678P	20010105 (60)
US 2000-179065P	20000131 (60)
US 2000-180628P	20000204 (60)
US 2000-214886P	20000628 (60)
US 2000-217487P	20000711 (60)
US 2000-225758P	20000814 (60)
US 2000-220963P	20000726 (60)
US 2000-217496P	20000711 (60)
US 2000-225447P	20000814 (60)
US 2000-218290P	20000714 (60)
US 2000-225757P	20000814 (60)
US 2000-226868P	20000822 (60)
US 2000-216647P	20000707 (60)

US	2000-225267P	20000814	(60)
US	2000-216880P	20000707	(60)
US	2000-225270P	20000814	(60)
US	2000-251869P	20001208	(60)
US	2000-235834P	20000927	(60)
US	2000-234274P	20000921	(60)
US	2000-234223P	20000921	(60)
US	2000-228924P	20000830	(60)
US	2000-224518P	20000814	(60)
US	2000-236369P	20000929	(60)
US	2000-224519P	20000814	(60)
US	2000-220964P	20000726	(60)
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US	2000-249299P	20001117	(60)
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US 2000-236368P	20000929 (60)
US 2000-251856P	20001208 (60)
US 2000-251868P	20001208 (60)
US 2000-229344P	20000901 (60)
US 2000-234997P	20000925 (60)
US 2000-229343P	20000901 (60)
US 2000-229345P	20000901 (60)
US 2000-229287P	20000901 (60)
US 2000-229513P	20000905 (60)
US 2000-231413P	20000908 (60)
US 2000-229509P	20000905 (60)
US 2000-236367P	20000929 (60)
US 2000-237039P	20001002 (60)
US 2000-237038P	20001002 (60)
US 2000-236370P	20000929 (60)
US 2000-236802P	20001002 (60)
US 2000-237037P	20001002 (60)
US 2000-237040P	20001002 (60)
US 2000-240960P	20001020 (60)
US 2000-239935P	20001013 (60)
US 2000-239937P	20001013 (60)
US 2000-241787P	20001020 (60)
US 2000-246474P	20001108 (60)
US 2000-246532P	20001108 (60)
US 2000-249216P	20001117 (60)
US 2000-249210P	20001117 (60)
US 2000-226681P	20000822 (60)
US 2000-225759P	20000814 (60)
US 2000-225213P	20000814 (60)
US 2000-227182P	20000822 (60)
US 2000-225214P	20000814 (60)
US 2000-235836P	20000927 (60)
US 2000-230438P	20000906 (60)
US 2000-215135P	20000630 (60)
US 2000-225266P	20000814 (60)
US 2000-249218P	20001117 (60)
US 2000-249208P	20001117 (60)
US 2000-249213P	20001117 (60)
US 2000-249212P	20001117 (60)
US 2000-249207P	20001117 (60)
US 2000-249245P	20001117 (60)
US 2000-249244P	20001117 (60)
US 2000-249217P	20001117 (60)
US 2000-249211P	20001117 (60)
US 2000-249215P	20001117 (60)
US 2000-249264P	20001117 (60)
US 2000-249214P	20001117 (60)
US 2000-249297P	20001117 (60)
US 2000-232400P	20000914 (60)
US 2000-231242P	20000908 (60)
US 2000-232081P	20000908 (60)
US 2000-232080P	20000908 (60)
US 2000-231414P	20000908 (60)
US 2000-231244P	20000908 (60)
US 2000-233064P	20000914 (60)
US 2000-233063P	20000914 (60)

US 2000-232397P	20000914 (60)
US 2000-232399P	20000914 (60)
US 2000-232401P	20000914 (60)
US 2000-241808P	20001020 (60)
US 2000-241826P	20001020 (60)
US 2000-241786P	20001020 (60)
US 2000-241221P	20001020 (60)
US 2000-246475P	20001108 (60)
US 2000-231243P	20000908 (60)
US 2000-233065P	20000914 (60)
US 2000-232398P	20000914 (60)
US 2000-234998P	20000925 (60)
US 2000-246477P	20001108 (60)
US 2000-246528P	20001108 (60)
US 2000-246525P	20001108 (60)
US 2000-246476P	20001108 (60)
US 2000-246526P	20001108 (60)
US 2000-249209P	20001117 (60)
US 2000-246527P	20001108 (60)
US 2000-246523P	20001108 (60)
US 2000-246524P	20001108 (60)
US 2000-246478P	20001108 (60)
US 2000-246609P	20001108 (60)
US 2000-246613P	20001108 (60)
US 2000-249300P	20001117 (60)
US 2000-249265P	20001117 (60)
US 2000-246610P	20001108 (60)
US 2000-246611P	20001108 (60)
US 2000-230437P	20000906 (60)
US 2000-251990P	20001208 (60)
US 2000-251988P	20001205 (60)
US 2000-251030P	20001205 (60)
US 2000-251479P	20001206 (60)
US 2000-256719P	20001205 (60)
US 2000-250160P	20001201 (60)
US 2000-251989P	20001208 (60)
US 2000-250391P	20001201 (60)
US 2000-254097P	20001211 (60)
US 2000-231968P	20000912 (60)
US 2000-226279P	20000818 (60)
US 2000-186350P	20000302 (60)
US 2000-184664P	20000224 (60)
US 2000-189874P	20000316 (60)
US 2000-198123P	20000418 (60)
US 2000-227009P	20000823 (60)
US 2000-235484P	20000926 (60)
US 2000-190076P	20000317 (60)
US 2000-209467P	20000607 (60)
US 2000-205515P	20000519 (60)
US 2001-259678P	20010105 (60)

DT Utility

FS APPLICATION

LREP HUMAN GENOME SCIENCES INC, 9410 KEY WEST AVENUE, ROCKVILLE, MD, 20850

CLMN Number of Claims: 24

ECL Exemplary Claim: 1

DRWN No Drawings

LN.CNT 24462

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention relates to novel ovarian related polynucleotides and the polypeptides encoded by these polynucleotides herein collectively known as "ovarian antigens," and the use of such ovarian antigens for detecting disorders of the ovaries and/or breast,

particularly the presence of ovarian and/or breast cancer and ovarian and/or breast cancer metastases. More specifically, isolated ovarian associated nucleic acid molecules are provided encoding novel ovarian associated polypeptides. Novel ovarian polypeptides and **antibodies** that bind to these polypeptides are provided. Also provided are vectors, host cells, and recombinant and synthetic methods for producing human ovarian associated polynucleotides and/or polypeptides. The invention further relates to diagnostic and therapeutic methods useful for diagnosing, treating, preventing and/or prognosing disorders related to the ovaries and/or breast, including ovarian and/or breast cancer, and therapeutic methods for treating such disorders. The invention further relates to screening methods for identifying agonists and antagonists of polynucleotides and polypeptides of the invention. The present invention further relates to methods and/or compositions for inhibiting the production and function of the polypeptides of the present invention.

L21 ANSWER 17 OF 44 USPATFULL  
AN 2002:84902 USPATFULL  
TI Nucleic acids, proteins and **antibodies**  
IN Rosen, Craig A., Laytonsville, MD, UNITED STATES  
Ruben, Steven M., Olney, MD, UNITED STATES  
PI US 2002044941 Al 20020418  
AI US 2001-925302 Al 20010810 (9)  
RLI Continuation-in-part of Ser. No. WO 2000-US5918, filed on 8 Mar 2000,  
UNKNOWN  
PRAI US 1999-124270P 19990312 (60)  
DT Utility  
FS APPLICATION  
LREP HUMAN GENOME SCIENCES INC, 9410 KEY WEST AVENUE, ROCKVILLE, MD, 20850  
CLMN Number of Claims: 23  
ECL Exemplary Claim: 1  
DRWN No Drawings  
LN.CNT 21121

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention relates to novel lung cancer related polynucleotides, the polypeptides encoded by these polynucleotides herein collectively referred to as "lung cancer antigens," and **antibodies** that immunospecifically bind these polypeptides, and the use of such lung cancer polynucleotides, antigens, and **antibodies** for detecting, treating, preventing and/or prognosing disorders of the lung, including, but not limited to, the presence of lung cancer and lung cancer metastases. More specifically, isolated lung cancer nucleic acid molecules are provided encoding novel lung cancer polypeptides. Novel lung cancer polypeptides and **antibodies** that bind to these polypeptides are provided. Also provided are vectors, host cells, and recombinant and synthetic methods for producing human lung cancer polynucleotides, polypeptides, and/or **antibodies**. The invention further relates to diagnostic and therapeutic methods useful for diagnosing, treating, preventing and/or prognosing disorders related to the lung, including lung cancer, and therapeutic methods for treating such disorders. The invention further relates to screening methods for identifying agonists and antagonists of polynucleotides and polypeptides of the invention. The invention further relates to methods and/or compositions for inhibiting or promoting the production and/or function of the polypeptides of the invention.

L21 ANSWER 18 OF 44 USPATFULL  
AN 2002:78729 USPATFULL  
TI Nucleic acids, proteins, and **antibodies**  
IN Rosen, Craig A., Laytonsville, MD, UNITED STATES  
Ruben, Steven M., Olney, MD, UNITED STATES

Barash, Steven C., Rockville, MD, UNITED STATES

PI US 2002042386 A1 20020411

AI US 2001-764870 A1 20010117 (9)

PRAI US 2000-179065P 20000131 (60)  
US 2000-180628P 20000204 (60)  
US 2000-214886P 20000628 (60)  
US 2000-217487P 20000711 (60)  
US 2000-225758P 20000814 (60)  
US 2000-220963P 20000726 (60)  
US 2000-217496P 20000711 (60)  
US 2000-225447P 20000814 (60)  
US 2000-218290P 20000714 (60)  
US 2000-225757P 20000814 (60)  
US 2000-226868P 20000822 (60)  
US 2000-216647P 20000707 (60)  
US 2000-225267P 20000814 (60)  
US 2000-216880P 20000707 (60)  
US 2000-225270P 20000814 (60)  
US 2000-251869P 20001208 (60)  
US 2000-235834P 20000927 (60)  
US 2000-234274P 20000921 (60)  
US 2000-234223P 20000921 (60)  
US 2000-228924P 20000830 (60)  
US 2000-224518P 20000814 (60)  
US 2000-236369P 20000929 (60)  
US 2000-224519P 20000814 (60)  
US 2000-220964P 20000726 (60)  
US 2000-241809P 20001020 (60)  
US 2000-249299P 20001117 (60)  
US 2000-236327P 20000929 (60)  
US 2000-241785P 20001020 (60)  
US 2000-244617P 20001101 (60)  
US 2000-225268P 20000814 (60)  
US 2000-236368P 20000929 (60)  
US 2000-251856P 20001208 (60)  
US 2000-251868P 20001208 (60)  
US 2000-229344P 20000901 (60)  
US 2000-234997P 20000925 (60)  
US 2000-229343P 20000901 (60)  
US 2000-229345P 20000901 (60)  
US 2000-229287P 20000901 (60)  
US 2000-229513P 20000905 (60)  
US 2000-231413P 20000908 (60)  
US 2000-229509P 20000905 (60)  
US 2000-236367P 20000929 (60)  
US 2000-237039P 20001002 (60)  
US 2000-237038P 20001002 (60)  
US 2000-236370P 20000929 (60)  
US 2000-236802P 20001002 (60)  
US 2000-237037P 20001002 (60)  
US 2000-237040P 20001002 (60)  
US 2000-240960P 20001020 (60)  
US 2000-239935P 20001013 (60)

DT Utility  
FS APPLICATION

LREP HUMAN GENOME SCIENCES INC, 9410 KEY WEST AVENUE, ROCKVILLE, MD, 20850  
CLMN Number of Claims: 24  
ECL Exemplary Claim: 1  
DRWN No Drawings  
LN.CNT 23133  
CAS INDEXING IS AVAILABLE FOR THIS PATENT.  
AB The present invention relates to novel proteins. More specifically,

isolated nucleic acid molecules are provided encoding novel polypeptides. Novel polypeptides and **antibodies** that bind to these polypeptides are provided. Also provided are vectors, host cells, and recombinant and synthetic methods for producing human polynucleotides and/or polypeptides, and **antibodies**. The invention further relates to diagnostic and therapeutic methods useful for diagnosing, treating, preventing and/or prognosing disorders related to these novel polypeptides. The invention further relates to screening methods for identifying agonists and antagonists of polynucleotides and polypeptides of the invention. The present invention further relates to methods and/or compositions for inhibiting or enhancing the production and function of the polypeptides of the present invention.

L21 ANSWER 19 OF 44 USPATFULL  
AN 2002:78442 USPATFULL  
TI Nucleic acids, proteins, and **antibodies**  
IN Rosen, Craig A., Laytonsville, MD, UNITED STATES  
Ruben, Steven M., Olney, MD, UNITED STATES  
Barash, Steven C., Rockville, MD, UNITED STATES  
PI US 2002042096 A1 20020411  
AI US 2001-764887 A1 20010117 (9)  
PRAI US 2000-179065P 20000131 (60)  
US 2000-180628P 20000204 (60)  
US 2000-214886P 20000628 (60)  
US 2000-217487P 20000711 (60)  
US 2000-225758P 20000814 (60)  
US 2000-220963P 20000726 (60)  
US 2000-217496P 20000711 (60)  
US 2000-225447P 20000814 (60)  
US 2000-218290P 20000714 (60)  
US 2000-225757P 20000814 (60)  
US 2000-226868P 20000822 (60)  
US 2000-216647P 20000707 (60)  
US 2000-225267P 20000814 (60)  
US 2000-216880P 20000707 (60)  
US 2000-225270P 20000814 (60)  
US 2000-251869P 20001208 (60)  
US 2000-235834P 20000927 (60)  
US 2000-234274P 20000921 (60)  
US 2000-234223P 20000921 (60)  
US 2000-228924P 20000830 (60)  
US 2000-224518P 20000814 (60)  
US 2000-236369P 20000929 (60)  
US 2000-224519P 20000814 (60)  
US 2000-220964P 20000726 (60)  
US 2000-241809P 20001020 (60)  
US 2000-249299P 20001117 (60)  
US 2000-236327P 20000929 (60)  
US 2000-241785P 20001020 (60)  
US 2000-244617P 20001101 (60)  
US 2000-225268P 20000814 (60)  
US 2000-236368P 20000929 (60)  
US 2000-251856P 20001208 (60)  
US 2000-251868P 20001208 (60)  
US 2000-229344P 20000901 (60)  
US 2000-234997P 20000925 (60)  
US 2000-229343P 20000901 (60)  
US 2000-229345P 20000901 (60)  
US 2000-229287P 20000901 (60)  
US 2000-229513P 20000905 (60)  
US 2000-231413P 20000908 (60)  
US 2000-229509P 20000905 (60)

US 2000-236367P 20000929 (60)  
US 2000-237039P 20001002 (60)  
US 2000-237038P 20001002 (60)  
US 2000-236370P 20000929 (60)  
US 2000-236802P 20001002 (60)  
US 2000-237037P 20001002 (60)  
US 2000-237040P 20001002 (60)  
US 2000-240960P 20001020 (60)  
US 2000-239935P 20001013 (60)

DT Utility

FS APPLICATION

LREP HUMAN GENOME SCIENCES INC, 9410 KEY WEST AVENUE, ROCKVILLE, MD, 20850

CLMN Number of Claims: 24

ECL Exemplary Claim: 1

DRWN No Drawings

LN.CNT 19583

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention relates to novel liver related polynucleotides and the polypeptides encoded by these polynucleotides herein collectively known as "liver antigens," and the use of such liver antigens for detecting disorders of the liver, particularly the presence of cancer of liver and cancer metastases. More specifically, isolated liver associated nucleic acid molecules are provided encoding novel liver associated polypeptides. Novel liver polypeptides and **antibodies** that bind to these polypeptides are provided. Also provided are vectors, host cells, and recombinant and synthetic methods for producing human liver associated polynucleotides and/or polypeptides. The invention further relates to diagnostic and therapeutic methods useful for diagnosing, treating, preventing and/or prognosing disorders related to the liver, including cancer of liver tissues, and therapeutic methods for treating such disorders. The invention further relates to screening methods for identifying agonists and antagonists of polynucleotides and polypeptides of the invention. The present invention further relates to methods and/or compositions for inhibiting the production and function of the polypeptides of the present invention.

L21 ANSWER 20 OF 44 USPATFULL

AN 2002:72627 USPATFULL

TI Nucleic, acids, proteins, and **antibodies**

IN Rosen, Craig A., Laytonsville, MD, UNITED STATES

Ruben, Steven M., Olney, MD, UNITED STATES

PI US 2002039764 A1 20020404

AI US 2001-925298 A1 20010810 (9)

RLI Continuation-in-part of Ser. No. WO 2000-US5881, filed on 8 Mar 2000, UNKNOWN

PRAI US 1999-124270P 19990312 (60)

DT Utility

FS APPLICATION

LREP HUMAN GENOME SCIENCES INC, 9410 KEY WEST AVENUE, ROCKVILLE, MD, 20850

CLMN Number of Claims: 23

ECL Exemplary Claim: 1

DRWN No Drawings

LN.CNT 20087

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention relates to novel ovarian cancer and/or breast cancer related polynucleotides, the polypeptides encoded by these polynucleotides herein collectively referred to as "ovarian and/or breast antigens," and **antibodies** that immunospecifically bind these polypeptides, and the use of such ovarian and/or breast polynucleotides, antigens, and **antibodies** for detecting, treating, preventing and/or prognosing disorders of the reproductive system, particularly disorders of the ovaries and/or breast, including,

but not limited to, the presence of ovarian and/or breast cancer and ovarian and/or breast cancer metastases. More specifically, isolated ovarian and/or breast nucleic acid molecules are provided encoding novel ovarian and/or breast polypeptides. Novel ovarian and/or breast polypeptides and **antibodies** that bind to these polypeptides are provided. Also provided are vectors, host cells, and recombinant and synthetic methods for producing human ovarian and/or breast polynucleotides, polypeptides, and/or **antibodies**. The invention further relates to diagnostic and therapeutic methods useful for diagnosing, treating, preventing and/or prognosing disorders related to the ovaries and/or breast, including ovarian and/or breast cancer, and therapeutic methods for treating such disorders. The invention further relates to screening methods for identifying agonists and antagonists of polynucleotides and polypeptides of the invention. The invention further relates to methods and/or compositions for inhibiting or promoting the production and/or function of the polypeptides of the invention.

L21 ANSWER 21 OF 44 USPATFULL  
AN 2002:72462 USPATFULL  
TI Methods of diagnosing and treating small intestinal bacterial overgrowth (SIBO) and SIBO-related conditions  
IN Lin, Henry C., Manhattan Beach, CA, UNITED STATES  
Pimentel, Mark, Los Angeles, CA, UNITED STATES  
PI US 2002039599 A1 20020404  
AI US 2001-837797 A1 20010417 (9)  
RLI Continuation-in-part of Ser. No. US 1999-374142, filed on 11 Aug 1999, PENDING Continuation-in-part of Ser. No. US 2000-546119, filed on 10 Apr 2000, PENDING Continuation-in-part of Ser. No. US 1999-420046, filed on 18 Oct 1999, PENDING Continuation-in-part of Ser. No. US 1999-359583, filed on 22 Jul 1999, ABANDONED Continuation of Ser. No. US 1997-832307, filed on 3 Apr 1997, GRANTED, Pat. No. US 5977175 Continuation of Ser. No. US 1995-442843, filed on 17 May 1995, ABANDONED  
DT Utility  
FS APPLICATION  
LREP SIDLEY & AUSTIN, 555 West Fifth Street, Los Angeles, CA, 90071-2909  
CLMN Number of Claims: 45  
ECL Exemplary Claim: 1  
DRWN 13 Drawing Page(s)  
LN.CNT 4226  
CAS INDEXING IS AVAILABLE FOR THIS PATENT.  
AB Disclosed is a method of treating small intestinal bacterial overgrowth (SIBO) or a SIBO-caused condition in a human subject. SIBO-caused conditions include irritable bowel syndrome, fibromyalgia, chronic pelvic pain syndrome, chronic fatigue syndrome, depression, impaired mentation, impaired memory, halitosis, tinnitus, sugar craving, autism, attention deficit/hyperactivity disorder, drug sensitivity, an autoimmune disease, and Crohn's disease. Also disclosed are a method of screening for the abnormally likely presence of SIBO in a human subject and a method of detecting SIBO in a human subject. A method of determining the relative severity of SIBO or a SIBO-caused condition in a human subject, in whom small intestinal bacterial overgrowth (SIBO) has been detected, is also disclosed.

L21 ANSWER 22 OF 44 USPATFULL  
AN 2002:67349 USPATFULL  
TI Coupling of peripheral tolerance to endogenous IL-10 promotes effective modulation of T cells and ameliorates autoimmune disease  
IN Zaghouani, Habib, Columbia, MO, UNITED STATES  
PI US 2002038002 A1 20020328  
AI US 2001-873901 A1 20010604 (9)  
PRAI US 2000-209527P 20000605 (60)

DT Utility  
FS APPLICATION  
LREP KNOBBE MARTENS OLSON & BEAR LLP, 620 NEWPORT CENTER DRIVE, SIXTEENTH FLOOR, NEWPORT BEACH, CA, 92660  
CLMN Number of Claims: 65  
ECL Exemplary Claim: 1  
DRWN 45 Drawing Page(s)  
LN.CNT 4140

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Immunomodulating agents comprising at least one Fc receptor ligand and at least one immunosuppressive factor are provided as are methods for their manufacture and use. The immunomodulating agents may be in the form of polypeptides or chimeric **antibodies** and preferably incorporate an immunosuppressive factor comprising a T cell receptor agonist or **antagonist**. The compounds and compositions of the invention may be used to selectively suppress the immune system to treat symptoms associated with immune disorders such as allergies, transplanted tissue rejection and autoimmune disorders including autoimmune diabetes, **rheumatoid arthritis** and multiple sclerosis.

L21 ANSWER 23 OF 44 USPATFULL  
AN 2002:48631 USPATFULL  
TI THERAPEUTIC COMPOUNDS FOR INHIBITING INTERLEUKIN-12 SIGNALING AND METHODS FOR USING SAME  
IN KLEIN, J. PETER, VASHON, WA, UNITED STATES  
KLAUS, STEPHEN J., SEATTLE, WA, UNITED STATES  
KUMAR, ANIL M., MERCER ISLAND, WA, UNITED STATES  
GONG, BAOQING, SHORELINE, WA, UNITED STATES  
PI US 2002028823 A1 20020307  
AI US 1999-288556 A1 19990409 (9)  
RLI Continuation-in-part of Ser. No. US 1998-8020, filed on 16 Jan 1998, ABANDONED  
DT Utility  
FS APPLICATION  
LREP McDERMOTT WILL & EMERY, 600 13TH STREET, N.W., WASHINGTON, DC, 20005-3096  
CLMN Number of Claims: 20  
ECL Exemplary Claim: 1  
DRWN 1 Drawing Page(s)  
LN.CNT 4381

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Novel heterocyclic compounds having a six membered ring structure fused to a five membered ring structure are found to be useful for the treatment and prevention of symptoms or manifestations associated with disorders affected by Interleukin-12 ("IL-12") intracellular signaling, such as, for example, Th1 cell-mediated disorders. The therapeutic compounds, pharmaceutically acceptable derivatives (e.g., resolved enantiomers, diastereomers, tautomers, salts and solvates thereof) or prodrugs thereof, have the following general formula: ##STR1##

Each X, Y and Z are independently selected from a member of the group consisting of C(R.<sub>sub.3</sub>), N, N(R.<sub>sub.3</sub>) and S. Each R.<sub>sub.1</sub>, R.<sub>sub.2</sub> and R.<sub>sub.3</sub> is substituted or unsubstituted and is independently selected from a member of the group consisting of hydrogen, halo, oxo, C.<sub>sub.1-20</sub>alkyl, C.<sub>sub.1-20</sub>hydroxyalkyl, C.<sub>sub.1-20</sub>thioalkyl, C.<sub>sub.1-20</sub>alkylamino, C.<sub>sub.1-20</sub>alkylaminoalkyl, C.<sub>sub.1-20</sub>aminoalkyl, C.<sub>sub.1-20</sub>aminoalkoxyalkenyl, C.<sub>sub.1-20</sub>aminoalkoxyalkynyl, C.<sub>sub.1-20</sub>diaminoalkyl, C.<sub>sub.1-20</sub>triaminoalkyl, C.<sub>sub.1-20</sub>tetraaminoalkyl, C.<sub>sub.5-15</sub>aminotrialkoxyamino, C.<sub>sub.1-20</sub>alkylamido,

C.sub.(1-20)alkylamidoalkyl, C.sub.(1-20)amidoalkyl,  
C.sub.(1-20)acetamidoalkyl, C.sub.(1-20)alkenyl, C.sub.(1-20)alkynyl,  
C.sub.(3-8)alkoxyl, C.sub.(1-11)alkoxyalkyl, and C.sub.(1-  
20)dialkoxyalkyl.

L21 ANSWER 24 OF 44 USPATFULL  
AN 2002:42953 USPATFULL  
TI Bispecific monoclonal **antibodies** to IL-12  
and IL-18  
IN Leung, Stewart, El Cerrito, CA, UNITED STATES  
Perez, H. Daniel, Kentfield, CA, UNITED STATES  
Miyamoto, Neil, Corte Madera, CA, UNITED STATES  
PA Schering AG, Berlin, GERMANY, FEDERAL REPUBLIC OF (U.S. corporation)  
PI US 2002025317 A1 20020228  
AI US 2001-907960 A1 20010719 (9)  
PRAI US 2000-219448P 20000720 (60)  
DT Utility  
FS APPLICATION  
LREP MILLEN, WHITE, ZELANO & BRANIGAN, P.C., 2200 CLARENDON BLVD., SUITE  
1400, ARLINGTON, VA, 22201  
CLMN Number of Claims: 30  
ECL Exemplary Claim: 1  
DRWN 7 Drawing Page(s)  
LN.CNT 1451  
CAS INDEXING IS AVAILABLE FOR THIS PATENT.  
AB A bispecific monoclonal **antibody** is described which comprises  
two moieties, one of which comprises an antigen-binding region which is  
specific for either the IL-12R. $\beta$ .1 or the IL-12R. $\beta$ .2 subunit of an  
IL-12 receptor, and the other of which comprises an  
antigen-binding region which is specific for either the IL-18R or the  
AcPL subunit of an IL-18 receptor.  
  
L21 ANSWER 25 OF 44 WPIDS (C) 2002 THOMSON DERWENT DUPLICATE 2  
AN 2001-244697 [25] WPIDS  
DNC C2001-073427  
TI Modulating responsiveness to a corticosteroid by administering a  
corticosteroid with an agent which antagonizes a target that regulates  
interferon-gamma production or an caspase family protease inhibitor,  
useful for treating asthma.  
DC B04 B05 D16  
IN BANERJEE, S; CARTER, A; GHAYUR, T; SEKUT, L; TRACEY, D E  
PA (BADI) BASF AG  
CYC 94  
PI WO 2001019373 A2 20010322 (200125)\* EN 152p  
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ  
NL OA PT SD SE SL SZ TZ UG ZW  
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CR CU CZ DE DK DM  
DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC  
LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE  
SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW  
AU 2000071276 A 20010417 (200140)  
ADT WO 2001019373 A2 WO 2000-US24725 20000908; AU 2000071276 A AU 2000-71276  
20000908  
FDT AU 2000071276 A Based on WO 200119373  
PRAI US 1999-398555 19990917  
AB WO 200119373 A UPAB: 20010508  
NOVELTY - A new method (M1) for modulating responsiveness to a  
corticosteroid in a subject comprises administering a corticosteroid with  
an agent (A1) which antagonizes a target that regulates production of  
interferon-gamma (IFN-gamma) or at least one agent (A2) that is an  
inhibitor of a caspase family protease.  
DETAILED DESCRIPTION - A method (M1) for modulating responsiveness to

a corticosteroid in a subject, comprising selecting a subject in need of modulation of responsiveness to a corticosteroid and administering:

(a) an agent (A1) which antagonizes a target that regulates production of interferon-gamma (IFN-gamma) in the subject, the agent being administered at a dosage and by a route sufficient to inhibit production of IFN-gamma; or

(b) at least one agent (A2) that is an inhibitor of a caspase family protease; and

(c) a corticosteroid.

The responsiveness of the subject to the corticosteroid is modulated as compared to when a corticosteroid alone is administered to the subject.

An INDEPENDENT CLAIM is also given for a method (M2) for regulating the production of IFN-gamma in a subject, comprising administering a corticosteroid and an agent which antagonizes a target that regulates production of IFN-gamma such that production of IFN-gamma is modulated in the subject.

ACTIVITY - Immunosuppressive; antiinflammatory; dermatological; antibacterial; cytostatic; antiasthmatic; anticonvulsant; antidiabetic; antiarthritic; antirheumatic; neuroprotective; antiallergic; antiulcer; ophthalmological; antianemic.

Interleukin converting enzyme (ICE)-deficient and wild type mice first were sensitized with Propionibacterium acnes cell wall material (1 mg per mouse) to induce low grade inflammation and six days later were challenged with lipopolysaccharide (LPS) (1 microgram per mouse in 0.1 ml of saline intravenously). Thirty minutes after LPS administration, the mice were treated with the corticosteroid dexamethasone (4 mg/kg per mouse in 0.5 ml 95% saline/0.5% ethanol, intraperitoneally). Control mice were treated with vehicle alone. All mice were bled 90 minutes after LPS administration and the serum samples were analyzed for the presence of tumor necrosis alpha (TNF-alpha) by standard ELISA (Enzyme linked immunosorbant assay).

Wild type and ICE deficient mice treated with vehicle alone had similar levels of serum TNF-alpha. Treatment of wild type mice with dexamethasone did not significantly affect serum TNF-alpha levels, demonstrating their resistance to steroid treatment in this septic shock model. In contrast, treatment of the ICE deficient mice with dexamethasone suppressed serum TNF-alpha levels by 74% (p less than 0.002). These data indicate that inhibition of ICE activity reverses resistance to steroid treatment in a septic shock model.

MECHANISM OF ACTION - IL-12 antagonist; IL-18 antagonist; phosphodiesterase IV inhibitor; a beta-2 agonist; a STAT4 inhibitor; an anti-IL-1-alpha antibody; an anti-IL-1-beta antibody; an anti-tumor necrosis factor antibody; a natural killer cell antagonist; a T-cell antagonist; caspase family protease inhibitor; gene therapy.

USE - The method is useful for treating a subject suffering from an autoimmune disease or disorder, an acute (e.g. infectious meningitis) or chronic (e.g. systemic lupus erythematosus or psoriasis) inflammatory disorder, septic shock or sepsis, graft versus host disease or transplant rejection, complications associated with post-surgical stress, Still's disease, leukemia or an immuno-inflammatory disease or disorder. The immuno-inflammatory disease or disorder is asthma, adult respiratory distress syndrome, systemic lupus erythematosus, inflammatory bowel disease, Crohn's disease, ulcerative colitis, multiple sclerosis, insulin-dependent diabetes mellitus, autoimmune arthritis, rheumatoid arthritis, juvenile rheumatoid arthritis, psoriatic arthritis, inflammatory pulmonary syndrome, pemphigus vulgaris, idiopathic thrombocytopenic purpura, autoimmune meningitis, myasthenia gravis, autoimmune thyroiditis, dermatitis, atopic dermatitis, eczematous dermatitis, psoriasis, Sjogren's Syndrome, keratoconjunctivitis sicca secondary to Sjogren's Syndrome, alopecia areata, allergic responses due to arthropod bite reactions, aphthous

ulcer, iritis, conjunctivitis, keratoconjunctivitis, cutaneous lupus erythematosus, scleroderma, vaginitis, proctitis, drug eruptions, Stevens-Johnson syndrome, leprosy reversal reactions, erythema nodosum leprosum, autoimmune uveitis, allergic encephalomyelitis, aplastic anemia, pure red cell anemia, idiopathic thrombocytopenia, polychondritis, Wegener's granulomatosis, chronic active hepatitis, Graves ophthalmopathy, primary biliary cirrhosis, uveitis posterior or interstitial lung fibrosis (claimed).

The method is useful for modulating corticosteroid responsiveness in a variety of clinical settings, for e.g. reversing steroid resistance, increasing steroid sensitivity, ameliorating a steroid rebound effect associated with administration of reduced dosages of the corticosteroid, or modulating corticosteroid activity, such that the corticosteroids can be tapered to zero (claimed).

Dwg.0/12

L21 ANSWER 26 OF 44 WPIDS (C) 2002 THOMSON DERWENT  
AN 2002-075345 [10] WPIDS  
DNC C2002-022528  
TI Use of apoptotic bodies and/or apoptotic cells for treatment and prophylaxis of T cell-mediated and inflammatory disorders such as psoriasis, atherosclerosis, diabetes and scleroderma in mammalian patients.  
DC B04 D16  
IN BOLTON, A E; MANDEL, A; SAUDER, D  
PA (VASO-N) VASOGEN IRELAND LTD; (BOLT-I) BOLTON A E; (MAND-I) MANDEL A; (SAUD-I) SAUDER D  
CYC 94  
PI WO 2001089536 A2 20011129 (200210)\* EN 20p  
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ  
NL OA PT SD SE SL SZ TR TZ UG ZW  
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CR CU CZ DE DK DM  
DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC  
LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE  
SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW  
CA 2309518 A1 20011125 (200210) EN  
AU 2001061986 A 20011203 (200221)  
US 2002051771 A1 20020502 (200234)  
ADT WO 2001089536 A2 WO 2001-CA758 20010525; CA 2309518 A1 CA 2000-2309518  
20000525; AU 2001061986 A AU 2001-61986 20010525; US 2002051771 A1 US  
2001-866488 20010525  
FDT AU 2001061986 A Based on WO 200189536  
PRAI CA 2000-2309518 20000525  
AB WO 200189536 A UPAB: 20020213  
NOVELTY - Use of apoptotic bodies and/or apoptotic cells for treatment and prophylaxis of T cell-mediated and inflammatory disorders, or in the preparation of a medicament for treatment and/or prophylaxis of T cell-mediated and inflammatory disorders in mammalian patients.  
ACTIVITY - Antipsoriatic; antirheumatic; antiarthritic;  
immunosuppressive; neuroprotective; antiallergic; dermatological;  
antiinflammatory; antidiabetic; antiatherosclerotic; antiasthmatic;  
antimicrobial. The effectiveness of treatment on contact hypersensitivity (CHS), an example of a Th-1-cell inflammatory disorder was assessed on laboratory mice. To induce CHS, the abdominal skin of each mouse was painted with dinitrofluorobenzene (DNFB). Apoptotic bodies were prepared from murine fibroblasts. The murine fibroblasts were treated with 50 nM sodium butyrate in RPMI (not defined) medium, at confluence for one day, and then the sodium butyrate medium was changed. To increase the number of apoptotic cells and bodies, the cells were additionally irradiated with ultra-violet (UV)-light. Supernatant containing floating cells was removed 24 hours following irradiation. Apoptotic bodies were quantitated. The pellet containing the apoptotic bodies was resuspended in phosphate

buffered saline (PBS). The cells to be stained for quantitation were resuspended in 1X binding buffer at a concentration of 1 multiply 10<sup>6</sup> cells/ml. Of the two groups of sensitized mice, the first, control group, received no treatment. The second, test group, was treated with an injection of suspended apoptotic bodies, 50 micro l volume containing at least 150000 bodies per injection of blood subjected to stressors. Treatments each involving intramuscular injection of 50 micro l of the respective liquid, started on the day of sensitization, and were repeated every day for a total of six days. On the same day as the last treatment, but after its administration, the animals were challenged with DNFB, by applying to the right ear to each animal 10 micro l of 0.2 % solution of DNFB in acetone and olive oil. To the left ear of each animal was applied the acetone/olive oil solvent without DNFB. Ear thickness was measured, 24 hours after challenge. The results were expressed as the thickness and difference in thickness of the right ears and the left ears of each animal, at 24 hours after challenge. The results showed a notable and significant reduction in ear thickness (inflammation) with the animals treated with the apoptotic cells and apoptotic bodies suspension, as compared with the untreated group, demonstrating a significant reduction in inflammation.

MECHANISM OF ACTION - Interleukin-10 (IL-10) agonist; tumor necrosis factor- gamma (TNF- gamma ), IL-6 and **IL-12 antagonist; vaccine.**

USE - The method is useful for treatment and/or prophylaxis of T cell-mediated and inflammatory disorders such as psoriasis, **rheumatoid arthritis**, scleroderma, lupus, diabetes mellitus, organ rejection, miscarriage, multiple sclerosis, inflammatory bowel disease, atherosclerosis and graft versus host disease in mammalian patients (claimed), and also microbial infections, asthma, contact hypersensitivity and other inflammatory allergic reactions. The method is also applicable to preconditioning against ingestions of poisons (poison ivy or poison oak reaction), exposure to toxic chemicals, radiation damage and exposure to air borne and water borne irritant substances, which cause damaging inflammation. In addition, it is applicable to inflammatory, allergic and T cell-mediated disorders of internal organs, such as kidney, liver, heart, etc.

Dwg.0/1

L21 ANSWER 27 OF 44 WPIDS (C) 2002 THOMSON DERWENT  
AN 2001-244560 [25] WPIDS  
DNC C2001-073385  
TI Composition comprising interleukin-12 p40 and IL-B30 polypeptide or its segment, useful for ameliorating **rheumatoid arthritis**, osteoarthritis, atherosclerosis, multiple sclerosis, vasculitis and tumor.  
DC B04 D16  
IN DE WAAL MALEYFT, R; KASTELEIN, R A; LIRA, S A; NARULA, S K; OPPMANN, B;  
RENNICK, D M; WIEKOWSKI, M T  
PA (SCHE) SCHERING CORP  
CYC 92  
PI WO 2001018051 A2 20010315 (200125)\* EN 69p  
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ  
NL OA PT SD SE SL SZ TZ UG ZW  
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CR CZ DE DK DM DZ  
EE ES FI GB GD GE HR HU ID IL IN IS JP KG KR KZ LC LK LR LT LU LV  
MA MD MG MK MN MX MZ NO NZ PL PT RO RU SE SG SI SK SL TJ TM TR TT  
TZ UA UZ VN YU ZA  
AU 2000073608 A 20010410 (200137)  
EP 1210434 A2 20020605 (200238) EN  
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT  
RO SE SI  
ADT WO 2001018051 A2 WO 2000-US24686 20000908; AU 2000073608 A AU 2000-73608  
20000908; EP 1210434 A2 EP 2000-961688 20000908, WO 2000-US24686 20000908

FDT AU 2000073608 A Based on WO 200118051; EP 1210434 A2 Based on WO 200118051  
PRAI US 1999-164616P 19991110; US 1999-393090 19990909  
AB WO 200118051 A UPAB: 20010508

NOVELTY - A composition (I) comprising a substantially pure polypeptide comprising a number of distinct segments of at least 7 contiguous amino acids from interleukin (IL)-12 p40 and/or IL-B30, and a substantially pure polypeptide comprising a segment of at least 11 contiguous amino acids from IL-12 p40 and/or IL-B30.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(1) an isolated or recombinant nucleic acid (II) encoding (I);  
(2) a cell (III) comprising (II);  
(3) a nucleic acid (IV) which hybridizes under wash conditions of 30 minutes at 50 deg. C and less than 1M salt to the natural mature coding portion of primate IL-12 p40 and IL-B30;

(4) an **antagonist** (V) of IL-12 p40/IL-B30 combined with a tumor necrosis factor-alpha (TNF alpha ) **antagonist**, an **IL-12 antagonist**, IL-10, or steroids;

(5) a binding compound (VI) comprising an antigen binding site from an **antibody**, which specifically binds to (I) and comprising a substantially pure polypeptide comprising IL-12 p40 and IL-B30 polypeptide, or a polypeptide comprising IL-12 p40 fused to IL-B30, but not to either IL-12 p40 or IL-B30 polypeptide;

(6) a kit (VII) comprising:  
(a) (I), and a compartment comprising the polypeptide, or instructions for use or disposal of reagents in the kit;  
(b) (II), and a compartment comprising (II), a compartment further comprising a primate IL-12 p40 or IL-B30, or instructions for use or disposal of reagents in the kit or (VI); and  
(c) a compartment comprising (VI), or instructions for use or disposal of reagents in the kit;

(7) producing (M1) an antigen:**antibody** complex, involves contacting, under appropriate conditions, a primate IL-12 p40/IL-B30 composition with (VI), allowing the complex to form;

(8) a composition (VIII) comprising (VI) which is sterile, or (VI) and a carrier such as an aqueous compound, including water, saline, and/or buffer;

(9) increasing (M2) the secretion of a primate IL-B30 involves expressing the polypeptide with IL-12 p40 or increasing the secretion of a primate IL-12 p40 involves expressing the IL-12 p40 with IL-B30; and

(10) screening (M3) for a receptor which binds (I) involves contacting the complex to a cell expressing the receptor under conditions allowing the complex to bind to the receptor, forming a detectable interaction.

ACTIVITY - Antirheumatic; antiarthritic; osteopathic; antiarthritic; neuroprotective; antiarteriosclerotic; cerebroprotective; vasotropic; cytostatic; antitumor; immunosuppressive.

MECHANISM OF ACTION - Modulator of physiology or development of cell in host; inducer of memory T-cell proliferation (claimed); modulator of trafficking or activation of leukocyte.

No supporting data is given.

USE - (I) is useful for modulating physiology or development of a cell or tissue in a host organism by contacting the cell with (I) or (V), resulting in an increased or decreased production of Interferon-gamma (IFN gamma ), an enhanced Th1 response such as anti-tumor effect, adjuvant effect, anti-viral effect or antagonized allergic effect, and amelioration of an autoimmune condition or a chronic inflammatory condition. The contacting is in combination with IL-18, IL-12, radiation therapy or chemotherapy, an immune adjuvant or an anti-viral

therapeutic. The **antagonist** is an **antibody** against **IL-12** receptor subunit beta 1. The **antagonist** or agonist of mammalian IL-B30 protein is useful for modulating the inflammatory response in an animal, by contacting cells in the animal with the agonist or **antagonist**, where the animal exhibits signs or symptoms of an acute phase inflammatory response in skin, lung, gastrointestinal, or liver tissue. The modulation is accelerating maturation of neutrophils into platelets and has an effect on immunoglobulin A and G (IgA and IgG). The **antagonist** is an **antibody** which binds to the mammalian IL-B30 or blocks signaling mediated by mammalian IL-B30. The **antagonist** or agonist is administered in combination with an anti-inflammatory cytokine agonist or **antagonist**, an analgesic, an anti-inflammatory agent, or a steroid. IL-B30 or its agonist is useful inducing the proliferation of memory T-cells (all claimed).

Agonist or **antagonist** of IL-B30 protein is useful for modulating the trafficking or activation of a leukocyte in an animal experiencing science or symptoms of autoimmunity, an inflammatory condition, tissue specific autoimmunity, degenerative autoimmunity, **rheumatoid arthritis**, osteoarthritis, atherosclerosis, multiple sclerosis, vasculitis, delayed hypersensitivities, skin grafting, a transplant, spinal injury, stroke, neurodegeneration, an infectious disease, ischemia, cancer, tumors, multiple myeloma, Castleman's disease, postmenopausal osteoporosis or IL-6-associated diseases.

**IL-12** p40/IL-B30 is useful as an immunogen for the production a antisera or **antibodies** specific for binding. (I) is useful for in vitro assays, scientific research, and the synthesis or manufacture of nucleic acids or **antibodies**. (II) is useful in forensic science.

Dwg.0/0

L21 ANSWER 28 OF 44 WPIDS (C) 2002 THOMSON DERWENT  
AN 2001-515399 [57] WPIDS  
DNC C2001-154207  
TI New substituted glutarimide derivatives are **IL-12 antagonists**, are useful as immunomodulators and for the treatment of angiopathy, hematological or oncological disorders.  
DC B03  
IN BUSCHMANN, H; FROSCH, S; GERMANN, T; WADE, E; ZIMMER, O  
PA (CHEF) GRUENENTHAL GMBH  
CYC 94  
PI DE 10002509 A1 20010726 (200157)\* 8p  
WO 2001053261 A1 20010726 (200157) DE  
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ  
NL OA PT SD SE SL SZ TR TZ UG ZW  
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CR CU CZ DE DK DM  
DZ EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC  
LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NO NZ PL PT RO RU SD SE  
SG SI SK SL TJ TM TR TT TZ UA UG US UZ VN YU ZA ZW  
AU 2001025140 A 20010731 (200171)  
ADT DE 10002509 A1 DE 2000-10002509 20000121; WO 2001053261 A1 WO 2001-EP155  
20010109; AU 2001025140 A AU 2001-25140 20010109  
FDT AU 2001025140 A Based on WO 200153261  
PRAI DE 2000-10002509 20000121  
AB DE 10002509 A UPAB: 20011005  
NOVELTY - Substituted glutarimide derivatives (I) are new.  
DETAILED DESCRIPTION - Substituted glutarimide derivatives of formula (I) are new.  
R1, R2 = COOR5, COR5 or CONR6R7, H, Cl, F, OH, NO2, NH2, 1-6C alkyl, 1-6C alkoxy, CHF2, CH2F, CF3 or an optionally substituted condensed benzol ring;  
R5 = 1-6C alkyl or 3-7C cycloalkyl;

R6, R7 = 1-6C alkyl, or R6+R7 together form pyrrolidine, piperidine, hexamethyleneimine, morpholine, thiomorpholine, piperazine or N-methylpiperazine;

R3 = H, OH or CH<sub>2</sub>NR6R7;

R4 = H, 1-3C alkyl, F, CF<sub>3</sub> or CHF<sub>2</sub>;

X = (CH<sub>2</sub>)<sub>n</sub>-NR<sub>8</sub>, (CH<sub>2</sub>)<sub>n</sub>-S, (CH<sub>2</sub>)<sub>q</sub>, C equivalent to C-(CH<sub>2</sub>)<sub>m</sub> or CH=CH-(CH<sub>2</sub>)<sub>m</sub>;

R8 = H, 1-6C alkyl, benzyl or phenylethyl (optionally substituted by Cl, F, CHF<sub>2</sub>, CH<sub>2</sub>F, CF<sub>3</sub>, OH, NO<sub>2</sub>, NH<sub>2</sub>, 1-6C alkyl or 1-6C alkoxy);

n = 1-3;

q = 2-4; and

m = 1 or 2.

Any available heteroatoms (on X) are bound to the glutarimide moiety and any methylene groups may be substituted by 1-3C alkyl, 1-3C alkoxy, F, Cl, CF<sub>3</sub> or OH. Provided that (a) R1 and R2 are not both H and (b) R1 and R2 cannot be OCH<sub>3</sub> when X = (CH<sub>2</sub>)<sub>n</sub>-S.

ACTIVITY - Immunomodulator; cytostatic; immunosuppressive; antiinflammatory; dermatological; antipsoriatic; antiasthmatic; antiulcer; hepatotropic; nephrotropic; antiallergic; antirheumatic; antiarthritic; neuroprotective; antidiabetic; antibacterial; antiarteriosclerotic; ophthalmological.

No biological data given.

MECHANISM OF ACTION - Interleukin-**antagonist-12**; HLA-**antagonist**.

USE - The compounds are useful as **IL-12** inhibitors with activity as immunomodulators and for the treatment of angiopathy, and hematological and oncological disorders (claimed). Non-claimed uses include the treatment of autoimmune disease, atopic dermatitis, psoriasis, eczema, bronchitis, pneumonia, bronchial asthma, ARDS, sarcoidosis, silicosis, fibrosis, gastroduodenal ulcers, Crohn's disease, ulcerative colitis, hepatitis, pancreatitis, appendicitis, peritonitis, nephritis, aphthosis, conjunctivitis, keratitis, uveitis, rhinitis, **rheumatoid arthritis**, HLA-B27 associated diseases, multiple sclerosis, juvenile onset diabetes, lupus erythematosus, sepsis, bacterial meningitis, cachexia, transplant rejection, graft-versus-host reactions, atherosclerosis, macular degeneration and diabetic retinopathy.

ADVANTAGE - Improved activity and stability against hydrolysis.  
Dwg. 0/0

L21 ANSWER 29 OF 44 BIOTECHDS COPYRIGHT 2002 THOMSON DERWENT AND ISI  
AN 2001-08257 BIOTECHDS  
TI Composition containing interleukin-12 p40 and IL-B30 protein or its segment, useful for ameliorating **rheumatoid arthritis**, osteoarthritis, atherosclerosis, multiple sclerosis, vasculitis and tumor;  
vector-mediated gene transfer and expression in host cell,  
**antibody and antagonist**  
AU Oppmann B; De Waal Malefyt R; Rennick D M; Kastelein R A; Wiekowski M T;  
Lira S A; Narula S K  
PA Schering-USA  
LO Kenilworth, NJ, USA.  
PI WO 2001018051 15 Mar 2001  
AI WO 2000-US24686 8 Sep 2000  
PRAI US 1999-164616 10 Nov 1999; US 1999-393090 9 Sep 1999  
DT Patent  
LA English  
OS WPI: 2001-244560 [25]  
AB A composition containing a substantially pure protein containing a number of distinct segments of at least 7 contiguous amino acids from interleukin (**IL**)-12 p40 and/or IL-B30, and a substantially pure protein containing a segment of at least 11 contiguous

amino acids from IL-12 p40 and/or IL-B30, is new. Also claimed are: a recombinant nucleic acid encoding the protein; a cell containing the nucleic acid; a nucleic acid which hybridizes under wash conditions of 30 min at 50 deg and less than 1M salt to the natural mature coding portion of primate IL-12 p40 and IL-B30; an antagonist of IL-12 p40/IL-B30 combined with a tumor necrosis factor-alpha (TNF-alpha) antagonist, an IL-12 antagonist, IL-10 or steroids; a binding compound containing an antigen binding site from an antibody which specifically binds to the protein; a kit containing the composition, polynucleotide and a binding compound; producing an antigen:antibody complex; a composition containing a binding compound; increasing the secretion of a primate IL-B30; and screening for a receptor which binds the composition. The composition is useful for modulating physiology or development of a cell or tissue0. (69pp)

L21 ANSWER 30 OF 44 USPATFULL  
AN 2001:221075 USPATFULL  
TI Retinoid antagonists and use thereof  
IN Bollag, Werner, Basel, Switzerland  
Klaus, Michael, Weil am Rhein, Germany, Federal Republic of  
Mohr, Peter, Basel, Switzerland  
Panina-Bordignon, Paola, Milan, Italy  
Rosenberger, Michael, Caldwell, NJ, United States  
Sinigaglia, Francesco, Milan, Italy  
PA Hoffman-La Roche Inc., Nutley, NJ, United States (U.S. corporation)  
PI US 6326397 B1 20011204  
AI US 1999-307009 19990507 (9)  
RLI Continuation-in-part of Ser. No. US 1998-189189, filed on 10 Nov 1998  
DT Utility  
FS GRANTED  
EXNAM Primary Examiner: Killos, Paul J.  
LREP Johnston, George W., Parise, John P.  
CLMN Number of Claims: 16  
ECL Exemplary Claim: 1  
DRWN 7 Drawing Figure(s); 5 Drawing Page(s)  
LN.CNT 1573  
CAS INDEXING IS AVAILABLE FOR THIS PATENT.  
AB The present invention relates to novel retinoid antagonists of the formula I ##STR1##

wherein the dotted bond can be either hydrogenated or form a double bond; and, when the dotted bond forms a double bond, R.<sup>1</sup> is lower alkyl and R.<sup>2</sup> is hydrogen; and, when the dotted bond is hydrogenated, R.<sup>1</sup> and R.<sup>2</sup> taken together are methylene to form a cis-substituted cyclopropyl ring; R.<sup>3</sup> is hydroxy or lower alkoxy; R.<sup>4</sup> is alkyl or alkoxy; and R.<sup>5</sup> and R.<sup>6</sup> are, independently, a C.<sub>4-12</sub> alkyl or a 5-12 cycloalkyl substituent containing from 1-3 rings which are either unsubstituted or substituted with from 1-3 lower alkyl groups, with the carbon atom of R.<sup>5</sup> and R.<sup>6</sup> being linked to the remainder of the molecule to form a quaternary carbon atom pharmaceutically acceptable salts of carbocyclic acids of the formula I; as well as method for the treatment of osteoporosis and preneoplastic and neoplastic diseases, and a method for reducing or abolishing adverse events in subjects receiving retinoid agonist treatment by administering a retinoid antagonist.

L21 ANSWER 31 OF 44 USPATFULL  
AN 2001:63494 USPATFULL  
TI Antibodies against human IL-12  
IN Gately, Maurice Kent, Parsippany, NJ, United States

PA Presky, David Howard, Glen Ridge, NJ, United States  
PI Hoffman-La Roche Inc., Nutley, NJ, United States (U.S. corporation)  
AI US 6225117 B1 20010501  
PRAI US 1999-232522 19990119 (9)  
DT Utility  
FS Granted  
EXNAM Primary Examiner: Chan, Christina Y.; Assistant Examiner: DiBrino, Marianne  
LREP Johnston, George W., Rocha-Tramaloni, Patricia S., Silverman, Robert A.  
CLMN Number of Claims: 23  
ECL Exemplary Claim: 1  
DRWN 7 Drawing Figure(s); 7 Drawing Page(s)  
LN.CNT 1122

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention relates to novel p75 heterodimer specific anti-human **IL-12 antibodies** that are characterized by a higher potency and greater efficacy in neutralizing human **IL-12** bioactivity than known heterodimer specific **IL-12 monoclonal antibodies**. The heterodimer specific **antibodies** recognize one or more **epitopes** of the human **IL-12** p75 heterodimer, but do not bind to the p40 subunit alone. The heterodimer specific **IL-12 antibodies** neutralize rhesus monkey **IL-12** bioactivity with a potency similar to their potency for neutralizing human **IL-12** bioactivity making them useful **IL-12 antagonists** for *in vivo* studies in the rhesus monkey.

L21 ANSWER 32 OF 44 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE  
3  
AN 2001:290669 BIOSIS  
DN PREV200100290669  
TI Antigen-specific T cell-mediated gene therapy in collagen-induced arthritis.  
AU Nakajima, Atsuo; Seroogy, Christine M.; Sandora, Matthew R.; Tarner, Ingo H.; Costa, Gina L.; Taylor-Edwards, Cariel; Bachmann, Michael H.; Contag, Christopher H.; Fathman, C. Garrison (1)  
CS (1) Department of Medicine, Division of Immunology and Rheumatology, School of Medicine, Stanford University, CCSR Building, Room 2225, Stanford, CA, 94305-5111: cfathman@leland.stanford.edu USA  
SO Journal of Clinical Investigation, (May, 2001) Vol. 107, No. 10, pp. 1293-1301. print.  
ISSN: 0021-9738.  
DT Article  
LA English  
SL English  
AB Autoantigen-specific T cells have tissue-specific homing properties, suggesting that these cells may be ideal vehicles for the local delivery of immunoregulatory molecules. We tested this hypothesis by using type II collagen-specific (CII-specific) CD4+ T hybridomas or primary CD4+ T cells after gene transfer, as vehicles to deliver an immunoregulatory protein for the treatment of collagen-induced arthritis (CIA), a mouse model of **rheumatoid arthritis (RA)**. CII-specific T cells or hybridomas were transduced using retroviral vectors to constitutively express the **IL-12 antagonist**, **IL-12 p40**. Transfer of engineered CD4+ T cells after immunization significantly inhibited the development of CIA, while cells transduced with vector control had no effect. The beneficial effect on CIA of **IL-12 p40**-transduced T cells required TCR specificity against CII, since transfer of T cells specific for another antigen producing equivalent amounts of **IL-12 p40** had

no effect. In vivo cell detection using bioluminescent labels and RT-PCR showed that transferred CII-reactive T-cell hybridomas accumulated in inflamed joints in mice with CIA. These results indicate that the local delivery of IL-12 p40 by T cells inhibited CIA by suppressing autoimmune responses at the site of inflammation. Modifying antigen-specific T cells by retroviral transduction for local expression of immunoregulatory proteins thus offers a promising strategy for treating RA.

L21 ANSWER 33 OF 44 USPATFULL  
AN 2000:138395 USPATFULL  
TI Treatment of T-helper cell type 2-mediated immune disease by retinoid antagonists  
IN Bollag, Werner, Basel, Switzerland  
Klaus, Michael, Weil am Rhein, Germany, Federal Republic of  
Panina-Bordignon, Paola, Milan, Italy  
Sinigaglia, Francesco, Milan, Italy  
PA Hoffmann-La Roche Inc., Nutley, NJ, United States (U.S. corporation)  
PI US 6133309 20001017  
AI US 1998-189189 19981110 (9)  
PRAI EP 1997-119776 19971112  
DT Utility  
FS Granted  
EXNAM Primary Examiner: Travers, Russell  
LREP Johnston, George W., Epstein, William H., Parise, John P.  
CLMN Number of Claims: 37  
ECL Exemplary Claim: 1  
DRWN No Drawings  
LN.CNT 780  
CAS INDEXING IS AVAILABLE FOR THIS PATENT.  
AB Retinoids with retinoid receptor antagonistic activity, pharmaceutically acceptable salts and pharmaceutically acceptable hydrolyzable esters thereof, have been found efficacious in treating T-helper cell type 2 (Th2)-mediated immune diseases, such as immunoglobulin E (IgE)-mediated allergic diseases.

L21 ANSWER 34 OF 44 USPATFULL  
AN 2000:50737 USPATFULL  
TI Methods and compositions for modulating responsiveness to corticosteroids  
IN Sekut, Les, Westborough, MA, United States  
Carter, Adam, Newburyport, MA, United States  
Ghayur, Tariq, Grafton, MA, United States  
Banerjee, Subhashis, Shrewsbury, MA, United States  
Tracey, Daniel E., Harvard, MA, United States  
PA BASF Aktiengesellschaft, Rheinland Pfalz, Germany, Federal Republic of (non-U.S. corporation)  
PI US 6054487 20000425  
AI US 1997-820692 19970318 (8)  
DT Utility  
FS Granted  
EXNAM Primary Examiner: Jarvis, William R. A.  
LREP Lahive & Cockfield, LLP  
CLMN Number of Claims: 46  
ECL Exemplary Claim: 1  
DRWN 3 Drawing Figure(s); 3 Drawing Page(s)  
LN.CNT 2404  
CAS INDEXING IS AVAILABLE FOR THIS PATENT.  
AB Method for modulating responsiveness to corticosteroids in a subject are provided. In the method of the invention, an agent which antagonizes a factor that regulates production of IFN-.gamma. in the subject is administered to the subject in combination with a corticosteroid such

that responsiveness of the subject to the corticosteroid is modulated as compared to when a corticosteroid alone is administered to the subject. In one embodiment, the agent is an interferon-.gamma. inducing factor (IGIF) **antagonist**. In another embodiment, the agent is an interleukin-12 (**IL-12**) **antagonist**. In a preferred embodiment, the agent is an inhibitor of a caspase family protease, preferably an ICE inhibitor. In another preferred embodiment, the agent is an anti-**IL-12** monoclonal **antibody**. Other preferred agents include phosphodiesterase IV inhibitors and beta-2 agonists. The methods of the invention can be used in the treatment of a variety of inflammatory and immunological diseases and disorders. Pharmaceutical compositions comprising an agent which antagonizes a factor that regulates production of IFN-.gamma. in a subject, a corticosteroid and a pharmaceutically acceptable carrier are also provided. A preferred composition comprises an ICE inhibitor, a corticosteroid and a pharmaceutically acceptable carrier.

L21 ANSWER 35 OF 44 CAPLUS COPYRIGHT 2002 ACS

AN 1999:487326 CAPLUS

DN 131:129052

TI **Antibodies** against human **IL-12**

IN Gately, Maurcie Kent; Presky, David Howard

PA F.Hoffmann-La Roche A.-G., Switz.

SO PCT Int. Appl., 47 pp.

CODEN: PIXXD2

DT Patent

LA English

FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9937682	A2	19990729	WO 1999-EP202	19990115
	W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, FI, GB, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
	RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
	AU 9925177	A1	19990115	AU 1999-25177	19990115
	CA 2318052	AA	19990729	CA 1999-2318052	19990115
	BR 9907743	A	20001017	BR 1999-7743	19990115
	EP 1049717	A2	20001108	EP 1999-904780	19990115
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, PT, IE, FI				
	JP 2002501085	T2	20020115	JP 2000-528602	19990115
	US 6225117	B1	20010501	US 1999-232522	19990119
	ZA 9900452	A	19990723	ZA 1999-452	19990121
PRAI	US 1998-72333P	P	19980123		
	WO 1999-EP202	W	19990115		

AB The present invention relates to p75 heterodimer specific anti-human **IL-12 antibodies** that are characterized by a higher potency and greater efficacy in neutralizing human **IL-12** bioactivity than known heterodimer specific **IL-12** monoclonal **antibodies**. The heterodimer specific **antibodies** recognize one or more **epitopes** of the human **IL-12** p75 heterodimer, but do not bind to the p40 subunit alone. The heterodimer specific **IL-12 antibodies** neutralize rhesus monkey **IL-12** bioactivity with a potency similar to their potency for neutralizing human **IL-12** bioactivity making them useful **IL-12** **antagonists**. The monoclonal **antibodies** are therefore useful for diseases assocd. with aberrant Th1-type helper cell

activity, e.g. multiple sclerosis, **rheumatoid arthritis**,  
autoimmune diabetes mellitus, Crohn's disease and ulcerative colitis.

L21 ANSWER 36 OF 44 USPATFULL  
AN 1999:43184 USPATFULL  
TI Membrane-bound cytokine compositions comprising GM-CSF and methods of modulating an immune response using same  
IN Hoo, William Soo, Carlsbad, CA, United States  
PA The Immune Response Corporation, Carlsbad, CA, United States (U.S. corporation)  
PI US 5891432 19990406  
AI US 1997-902516 19970729 (8)  
DT Utility  
FS Granted  
EXNAM Primary Examiner: Spector, Lorraine  
LREP Campbell & Flores LLP  
CLMN Number of Claims: 24  
ECL Exemplary Claim: 1,13  
DRWN 9 Drawing Figure(s); 7 Drawing Page(s)  
LN.CNT 1917  
CAS INDEXING IS AVAILABLE FOR THIS PATENT.  
AB The present invention provides a cellular vaccine having a membrane-bound fusion protein that includes a non-**antibody** immunomodulatory molecule such as GM-CSF operatively fused to a heterologous membrane attachment domain. Non-**antibody** immunomodulatory molecules useful in the invention include immunostimulatory and immunosuppressive molecules such as cytokines. In one embodiment, the invention provides a cellular vaccine having a membrane-bound fusion protein that includes a non-**antibody** immunomodulatory molecule operatively fused to a heterologous membrane attachment domain and, additionally, a disease-associated antigen or immunogenic **epitope** thereof. Further provided by the invention are methods of modulating an immune response against a disease-associated antigen by administering to an individual a cellular vaccine having a membrane-bound fusion protein that includes a non-**antibody** immunomodulatory molecule operatively fused to a heterologous membrane attachment domain.

L21 ANSWER 37 OF 44 WPIDS (C) 2002 THOMSON DERWENT DUPLICATE 4  
AN 1998-520957 [44] WPIDS  
DNC C1998-156445  
TI Modulating responsiveness to corticosteroid e.g. in treating auto-immune diseases - by administering agent antagonising target that regulates production of interferon gamma.  
DC B01 B04 B05  
IN BANERJEE, S; CARTER, A; GHAYUR, T; SEKUT, L; TRACEY, D E  
PA (BADI) BASF AG  
CYC 81  
PI WO 9841232 A2 19980924 (199844)\* EN 112p  
RW: AT BE CH DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL OA  
PT SD SE SZ UG ZW  
W: AL AM AT AU AZ BA BB BG BR BY CA CH CN CZ DE DK EE ES FI GB GE GH  
GM GW HU ID IL IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK  
MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT UA UG US  
UZ VN YU ZW  
AU 9867604 A 19981012 (199907)  
NO 9904506 A 19991117 (200005)  
CZ 9903127 A3 20000315 (200021)  
EP 998300 A1 20000510 (200027) EN  
R: AT BE CH DE DK ES FI FR GB GR IE IT LI LU NL PT SE  
US 6054487 A 20000425 (200027)  
ES 2146192 T1 20000801 (200040)

BR 9810409 A 20000822 (200050)  
CN 1269722 A 20001011 (200103)  
SK 9901221 A3 20001211 (200103)  
MX 9908433 A1 19991201 (200110)  
KR 2000076420 A 20001226 (200134)  
AU 734756 B 20010621 (200141)  
JP 2002504091 W 20020205 (200212) 154p  
HU 2001004439 A2 20020429 (200238)

ADT WO 9841232 A2 WO 1998-US4916 19980312; AU 9867604 A AU 1998-67604 19980312; NO 9904506 A WO 1998-US4916 19980312, NO 1999-4506 19990917; CZ 9903127 A3 WO 1998-US4916 19980312, CZ 1999-3127 19980312; EP 998300 A1 EP 1998-912929 19980312, WO 1998-US4916 19980312; US 6054487 A US 1997-820692 19970318; ES 2146192 T1 EP 1998-912929 19980312; BR 9810409 A BR 1998-10409 19980312, WO 1998-US4916 19980312; CN 1269722 A CN 1998-805124 19980312; SK 9901221 A3 WO 1998-US4916 19980312, SK 1999-1221 19980312; MX 9908433 A1 MX 1999-8433 19990914; KR 2000076420 A WO 1998-US4916 19980312, KR 1999-708524 19990918; AU 734756 B AU 1998-67604 19980312; JP 2002504091 W JP 1998-540633 19980312, WO 1998-US4916 19980312; HU 2001004439 A2 WO 1998-US4916 19980312, HU 2001-4439 19980312

FDT AU 9867604 A Based on WO 9841232; CZ 9903127 A3 Based on WO 9841232; EP 998300 A1 Based on WO 9841232; ES 2146192 T1 Based on EP 998300; BR 9810409 A Based on WO 9841232; KR 2000076420 A Based on WO 9841232; AU 734756 B Previous Publ. AU 9867604, Based on WO 9841232; JP 2002504091 W Based on WO 9841232; HU 2001004439 A2 Based on WO 9841232

PRAI US 1998-16346 19980130; US 1997-820692 19970318

AB WO 9841232 A UPAB: 19981104  
Modulating responsiveness to corticosteroids comprises administering: (a) an agent which antagonises a target that regulates production of interferon- gamma (IFN- gamma), to inhibit production of IFN- gamma and (b) a corticosteroid.  
Preferably, the agent which antagonises a target that regulates production of IFN- gamma is an IL-18 **antagonist** e.g. an inhibitor of a caspase family protease (especially an ICE inhibitor) or an **antibody (fragment)** or engineered binding protein that binds IL-18 or an IL-18 receptor. The agent may also be an **IL-12 antagonist** e.g. an agent that stimulates cyclic AMP production in cells that produce **IL-12**, especially a phosphodiesterase IV inhibitor such as a 4-arylpiperidinone, rolipram, denbufylline, tibenelast, nitraquazone, CP-80633, CP-77059 or a quinazolinedione or a beta -2 agonist such as salmeterol, fenoterol or isoproterenol.  
USE- The process is used for treating septic shock, Crohn's disease, asthma, graft versus host disease or transplant rejection autoimmune disease or disorder and immunoinflammatory diseases or disorders comprising adult respiratory distress syndrome, systemic lupus erythematosus, inflammatory bowel disease, ulcerative colitis, multiple sclerosis, insulin dependent diabetes mellitus, **rheumatoid arthritis**, **juvenile rheumatoid arthritis**, psoriatic arthritis, inflammatory pulmonary syndrome, pemphigus vulgaris, idiopathic thrombocytopenic purpura, autoimmune meningitis, myasthenia gravis, autoimmune thyroiditis, dermatitis, atopic dermatitis, eczematous dermatitis, psoriasis, Sjogren's syndrome, keratoconjunctivitis, cutaneous lupus erythematosus, scleroderma, vaginitis, proctitis, drug eruptions, Stevens-Johnson syndrome, leprosy reversal reactions, erythema nodosum leprosum, autoimmune uveitis, allergic encephalomyelitis, aplastic anaemia, pure red cell anaemia, idiopathic thrombocytopenia, polychondritis, Wegener's granulomatosis, chronic active hepatitis, Graves ophthalmopathy, primary biliary cirrhosis, uveitis posterior and interstitial lung fibrosis. Administration is oral, intravenous or ophthalmic.  
ADVANTAGE - The process reverses steroid resistance and increases steroid sensitivity.

AB The present invention relates to a novel **antibody** against the **IL-12** receptor and a novel combination of antibodies against the **IL-12** receptor. The novel anti-**IL-12** receptor antibody, designated as 2B10, provided in accordance with the present invention binds to the human **IL-12** receptor but which is not capable of inhibiting the binding of human **IL-12** to the high affinity human **IL-12** receptor and is not capable of neutralizing human **IL-12** bioactivity by binding to human **IL-12** receptor.

L21 ANSWER 40 OF 44 USPATFULL  
AN 1998:135151 USPATFULL  
TI Human receptor for interleukin-12  
IN Chua, Anne On, Wayne, NJ, United States  
Gubler, Ulrich Andreas, Glen Ridge, NJ, United States  
PA Hoffmann-La Roche Inc., Nutley, NJ, United States (U.S. corporation)  
PI US 5831007 19981103  
AI US 1995-419652 19950411 (8)  
RLI Division of Ser. No. US 1994-248532, filed on 31 May 1994, now patented, Pat. No. US 5536657 which is a continuation-in-part of Ser. No. US 1993-94713, filed on 19 Jul 1993, now abandoned  
DT Utility  
FS Granted  
EXNAM Primary Examiner: Ulm, John  
LREP Johnston, George W., Epstein, William H., Bucholz, Briana C.  
CLMN Number of Claims: 10  
ECL Exemplary Claim: 1  
DRWN 35 Drawing Figure(s); 26 Drawing Page(s)  
LN.CNT 1937  
CAS INDEXING IS AVAILABLE FOR THIS PATENT.  
AB This invention relates to substantially pure Interleukin-12 receptor cDNAs and protein and uses therefore. The Interleukin-12 receptor is shown to be a member of the cytokine receptor superfamily and has a high homology to human gp130.

L21 ANSWER 41 OF 44 WPIDS (C) 2002 THOMSON DERWENT  
AN 1997-147515 [14] WPIDS  
DNN N1997-122015 DNC C1997-047130  
TI New interleukin-12 beta-2 receptor and high binding affinity complexes - have a high affinity for interleukin-12, and are used to treat auto immune diseases.  
DC B04 D16 S03  
IN GUBLER, U A; PRESKY, D H  
PA (HOFF) HOFFMANN LA ROCHE & CO AG F; (HOFF) HOFFMANN LA ROCHE INC  
CYC 20  
PI EP 759466 A2 19970226 (199714)\* EN 53p  
R: AT BE CH DE DK ES FI FR GB GR IE IT LI LU MC NL PT SE  
JP 09132598 A 19970520 (199730) 43p  
EP 759466 A3 19970528 (199732)  
US 5840530 A 19981124 (199903)  
US 5852176 A 19981222 (199907) #  
US 5919903 A 19990706 (199933)  
JP 2948150 B2 19990913 (199943) 43p  
ADT EP 759466 A2 EP 1996-111807 19960723; JP 09132598 A JP 1996-196385 19960725; EP 759466 A3 EP 1996-111807 19960723; US 5840530 A Provisional US 1995-1701P 19950801, Provisional US 1996-18674P 19960530, US 1996-685118 19960723; US 5852176 A Div ex US 1996-685118 19960723, US 1997-915495 19970820; US 5919903 A Provisional US 1995-1701P 19950801, Provisional US 1996-18674P 19960530, Div ex US 1996-685118 19960723, US 1997-914520 19970819; JP 2948150 B2 JP 1996-196385 19960725  
FDT JP 2948150 B2 Previous Publ. JP 09132598

PRAI US 1996-18674P 19960530; US 1995-1701P 19950801; US 1996-685118  
19960723; US 1997-915495 19970820; US 1997-914520 19970819  
AB EP 759466 A UPAB: 19970407  
A novel low binding affinity (BA) interleukin-12 (**IL-12**) beta 2 receptor protein (A), or a **fragment**, has a low BA for **IL-12**, and when complexed with an **IL-12** beta 1 receptor protein (B), forms a complex having a high BA for **IL-12**. Also new are: (1) a complex with a high BA for **IL-12**, comprising (A) or a **fragment**, complexed with **IL-12** beta 1 receptor protein, or a **fragment**, which has a low BA for **IL-12**, and, when complexed with (A), has a high BA for **IL-12**; (2) a protein encoded by first and second nucleic acids, the first comprising two subsequences (SS), where one SS encodes a soluble **fragment** of (A), and the other SS (SS2) encodes all the domains of the constant region of the heavy chain of human Ig, except the first domain of the constant region, and the second nucleic acid has two SS, where one SS encodes a soluble **fragment** of (B) and the other SS is as for SS2; (3) nucleic acids encoding the proteins or complexes; (4) vectors contg. the nucleic acid of (3); (5) host cells transformed with the nucleic acid of (3); and (6) **antibodies** against (A) or (B).  
USE - The proteins, complexes or **antibodies** may be used in therapeutic compsns., pref. with at least 1 cytokine antagonists (claimed). The compsns. are used to treat autoimmune dysfunctions (claimed), such as **rheumatoid arthritis**, inflammatory bowel disease and multiple sclerosis. The proteins or complexes can also be used to detect **antagonists** and agonists of **IL-12** activity (claimed).  
Dwg. 0/6

L21 ANSWER 42 OF 44 USPATFULL  
AN 96:63048 USPATFULL  
TI Recombinant DNA encoding human receptor for interleukin-12  
IN Chua, Anne O., Wayne, NJ, United States  
Gubler, Ulrich A., Glen Ridge, NJ, United States  
PA Hoffmann-La Roche Inc., Nutley, NJ, United States (U.S. corporation)  
PI US 5536657 19960716  
AI US 1994-248532 19940531 (8)  
RLI Continuation-in-part of Ser. No. US 1993-94713, filed on 19 Jul 1993,  
now abandoned  
DT Utility  
FS Granted  
EXNAM Primary Examiner: Ulm, John  
LREP Gould, George M., Johnston, George W., Kass, Alan P.  
CLMN Number of Claims: 10  
ECL Exemplary Claim: 1  
DRWN 34 Drawing Figure(s); 25 Drawing Page(s)  
LN.CNT 1755  
CAS INDEXING IS AVAILABLE FOR THIS PATENT.  
AB This invention relates to substantially pure Interleukin-12 receptor cDNAs and protein and uses therefore. The Interleukin-12 receptor is shown to be a member of the cytokine receptor superfamily and has a high homology to human gp130.

L21 ANSWER 43 OF 44 CAPLUS COPYRIGHT 2002 ACS  
AN 1995:934127 CAPLUS  
DN 123:337469  
TI Use of **IL-12** and **IL-12** **antagonists** in treatment of autoimmune diseases  
IN Leonard, John P.; Goldman, Samuel; O'Hara, Richard, Jr.  
PA Genetics Institute, Inc., USA  
SO PCT Int. Appl., 37 pp.

CODEN: PIXXD2

DT Patent

LA English

FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9524918	A1	19950921	WO 1995-US2550	19950307
	W: AU, CA, JP RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
	ZA 9500960	A	19951010	ZA 1995-960	19950207
	TW 400233	B	20000801	TW 1995-84101380	19950214
	IL 112677	A1	20000131	IL 1995-112677	19950216
	CA 2185565	AA	19950921	CA 1995-2185565	19950307
	AU 9519749	A1	19951003	AU 1995-19749	19950307
	AU 689236	B2	19980326		
	EP 750509	A1	19970102	EP 1995-912666	19950307
	EP 750509	B1	20020515		
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE JP 09510444	T2	19971021	JP 1995-524044	19950307
	EP 1179348	A2	20020213	EP 2001-117762	19950307
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE US 6338848	B1	20020115	US 2000-513380	20000225
PRAI	US 1994-212629	A	19940314		
	EP 1995-912666	A3	19950307		
	WO 1995-US2550	W	19950307		
	US 1995-560943	B1	19951120		
AB	Autoimmune conditions such as multiple sclerosis, systemic lupus erythematosus, <b>rheumatoid arthritis</b> , autoimmune pulmonary inflammation, Guillain-Barre syndrome, autoimmune thyroiditis, insulin-dependent diabetes mellitus, and autoimmune inflammatory eye disease, esp. conditions which are promoted by an increase in levels of IFN-.gamma. or TNF-.alpha., are treated in mammals by administering <b>IL-12</b> or an <b>IL-12 antagonist</b> . Thus, lymphocytes from mice immunized with myelin proteolipid protein, and restimulated with a synthetic peptide from this protein, were injected into naive mice. The injected mice developed exptl. allergic encephalomyelitis which was exacerbated by incubation of these lymphocytes with <b>IL-12</b> during restimulation, and alleviated by injection of a polyclonal <b>antibody to IL-12</b> .				
L21	ANSWER 44 OF 44	MEDLINE			
AN	94032770	MEDLINE			
DN	94032770	PubMed ID: 8218599			
TI	Clinical and preclinical studies presented at the Keystone Symposium on Arthritis, Related Diseases, and Cytokines.				
AU	Ralph P				
CS	Department of Immunology, Genentech, Inc., South San Francisco, CA 94080.				
SO	LYMPHOKINE AND CYTOKINE RESEARCH, (1993 Aug) 12 (4) 261-3. Journal code: 9107882. ISSN: 1056-5477.				
CY	United States				
DT	Conference; Conference Article; (CONGRESSES)				
LA	English				
FS	Priority Journals				
EM	199312				
ED	Entered STN: 19940117 Last Updated on STN: 19990129 Entered Medline: 19931203				
AB	Topics include treatment of multiple sclerosis (MS) with T cell receptor (TCR) peptides, <b>rheumatoid arthritis (RA)</b> with IL-1ra, IL-2 toxin conjugate, or <b>antibodies</b> to TNF, to CD4, or to ICAM-1, sepsis and five other diseases with IL-1ra, and treatment of experimental animal diseases with soluble receptors, <b>IL-</b>				

**12, TGF-beta2, or small molecule antagonists of cytokines.**

=> d clm 1 14

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L21 ANSWER 1 OF 44 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.DUPLICATE  
1

AN 2002:166945 BIOSIS

DN PREV200200166945

TI Use of **IL-12** and **IL-12**

**antagonists** in the treatment of autoimmune diseases.

AU Leonard, John (1); Goldman, Samuel; O'Hara, Richard, Jr.

CS (1) Auburn, NH USA

ASSIGNEE: Genetics Institute, Inc.

PI US 6338848 January 15, 2002

SO Official Gazette of the United States Patent and Trademark Office Patents, (Jan. 15, 2002) Vol. 1254, No. 3, pp. No Pagination.

<http://www.uspto.gov/web/menu/patdata.html>. e-file.

ISSN: 0098-1133.

DT Patent

LA English

L21 ANSWER 14 OF 44 USPATFULL

AN 2002:106416 USPATFULL

TI Nucleic acids, proteins and **antibodies**

IN Rosen, Craig A., Laytonsville, MD, UNITED STATES  
Ruben, Steven M., Olney, MD, UNITED STATES

PI US 2002055627 A1 20020509

AI US 2001-925299 A1 20010810 (9)

RLI Continuation of Ser. No. WO 2000-US5883, filed on 8 Mar 2000, UNKNOWN

PRAI US 1999-124270P 19990312 (60)

DT Utility

FS APPLICATION

LN.CNT 20658

INCL INCLM: 536/023.500

INCLS: 435/320.100; 435/325.000; 530/324.000; 530/387.900; 514/002.000;  
435/007.200

NCL NCLM: 536/023.500

NCLS: 435/320.100; 435/325.000; 530/324.000; 530/387.900; 514/002.000;  
435/007.200

IC [7]

ICM: A01N037-18

ICS: A61K038-00; G01N033-53; G01N033-567; C07H021-04; C12N015-00;  
C12N015-09; C12N015-63; C12N015-70; C12N015-74; C07K005-00; C07K007-00;  
C07K016-00; C07K017-00; C12N005-00; C12N005-02; C12P021-08

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

=> d clm 14

L21 ANSWER 14 OF 44 USPATFULL

CLM What is claimed is:

1. An isolated nucleic acid molecule comprising a polynucleotide having a nucleotide sequence at least 95% identical to a sequence selected from the group consisting of: (a) a polynucleotide **fragment** of SEQ

ID NO:X or a polynucleotide **fragment** of the cDNA sequence included in the related cDNA clone, which is hybridizable to SEQ ID NO:X; (b) a polynucleotide encoding a polypeptide **fragment** of SEQ ID NO:Y or a polypeptide **fragment** encoded by the cDNA sequence included in the related cDNA clone, which is hybridizable to SEQ ID NO:X; (c) a polynucleotide encoding a polypeptide **fragment** of a polypeptide encoded by SEQ ID NO:X or a polypeptide **fragment** encoded by the cDNA sequence included in the related cDNA clone, which is hybridizable to SEQ ID NO:X; (d) a polynucleotide encoding a polypeptide domain of SEQ ID NO:Y or a polypeptide domain encoded by the cDNA sequence included in the related cDNA clone, which is hybridizable to SEQ ID NO:X; (e) a polynucleotide encoding a polypeptide **epitope** of SEQ ID NO:Y or a polypeptide **epitope** encoded by the cDNA sequence included in the related cDNA clone, which is hybridizable to SEQ ID NO:X; (f) a polynucleotide encoding a polypeptide of SEQ ID NO:Y or the cDNA sequence included in the related cDNA clone, which is hybridizable to SEQ ID NO:X, having biological activity; (g) a polynucleotide which is a variant of SEQ ID NO:X; (h) a polynucleotide which is an allelic variant of SEQ ID NO:X; (i) a polynucleotide which encodes a species homologue of the SEQ ID NO:Y; (j) a polynucleotide capable of hybridizing under stringent conditions to any one of the polynucleotides specified in (a)-(i), wherein said polynucleotide does not hybridize under stringent conditions to a nucleic acid molecule having a nucleotide sequence of only A residues or of only T residues.

2. The isolated nucleic acid molecule of claim 1, wherein the polynucleotide **fragment** comprises a nucleotide sequence encoding a protein.

3. The isolated nucleic acid molecule of claim 1, wherein the polynucleotide **fragment** comprises a nucleotide sequence encoding the sequence identified as SEQ ID NO:Y or the polypeptide encoded by the cDNA sequence included in the related cDNA clone, which is hybridizable to SEQ ID NO:X.

4. The isolated nucleic acid molecule of claim 1, wherein the polynucleotide **fragment** comprises the entire nucleotide sequence of SEQ ID NO:X or the cDNA sequence included in the related cDNA clone, which is hybridizable to SEQ ID NO:X.

5. The isolated nucleic acid molecule of claim 2, wherein the nucleotide sequence comprises sequential nucleotide deletions from either the C-terminus or the N-terminus.

6. The isolated nucleic acid molecule of claim 3, wherein the nucleotide sequence comprises sequential nucleotide deletions from either the C-terminus or the N-terminus.

7. A recombinant vector comprising the isolated nucleic acid molecule of claim 1.

8. A method of making a recombinant host cell comprising the isolated nucleic acid molecule of claim 1.

9. A recombinant host cell produced by the method of claim 8.

10. The recombinant host cell of claim 9 comprising vector sequences.

11. An isolated polypeptide comprising an amino acid sequence at least 95% identical to a sequence selected from the group consisting of: (a) a polypeptide **fragment** of SEQ ID NO:Y or of the sequence

encoded by the cDNA included in the related cDNA clone; (b) a polypeptide **fragment** of SEQ ID NO:Y or of the sequence encoded by the cDNA included in the related cDNA clone, having biological activity; (c) a polypeptide domain of SEQ ID NO:Y or of the sequence encoded by the cDNA included in the related cDNA clone; (d) a polypeptide **epitope** of SEQ ID NO:Y or of the sequence encoded by the cDNA included in the related cDNA clone; (e) a full length protein of SEQ ID NO:Y or of the sequence encoded by the cDNA included in the related cDNA clone; (f) a variant of SEQ ID NO:Y; (g) an allelic variant of SEQ ID NO:Y; or (h) a species homologue of the SEQ ID NO:Y.

12. The isolated polypeptide of claim 11, wherein the full length protein comprises sequential amino acid deletions from either the C-terminus or the N-terminus.
13. An isolated **antibody** that binds specifically to the isolated polypeptide of claim 11.
14. A recombinant host cell that expresses the isolated polypeptide of claim 11.
15. A method of making an isolated polypeptide comprising: (a) culturing the recombinant host cell of claim 14 under conditions such that said polypeptide is expressed; and (b) recovering said polypeptide.
16. The polypeptide produced by claim 15.
17. A method for preventing, treating, or ameliorating a medical condition, comprising administering to a mammalian subject a therapeutically effective amount of the polypeptide of claim 11 or the polynucleotide of claim 1.
18. A method of diagnosing a pathological condition or a susceptibility to a pathological condition in a subject comprising: (a) determining the presence or absence of a mutation in the polynucleotide of claim 1; and (b) diagnosing a pathological condition or a susceptibility to a pathological condition based on the presence or absence of said mutation.
19. A method of diagnosing a pathological condition or a susceptibility to a pathological condition in a subject comprising: (a) determining the presence or amount of expression of the polypeptide of claim 1 in a biological sample; and (b) diagnosing a pathological condition or a susceptibility to a pathological condition based on the presence or amount of expression of the polypeptide.
20. A method for identifying a binding partner to the polypeptide of claim 1 comprising: (a) contacting the polypeptide of claim 11 with a binding partner; and (b) determining whether the binding partner effects an activity of the polypeptide.
21. The gene corresponding to the cDNA sequence of SEQ ID NO:Y.
22. A method of identifying an activity in a biological assay, wherein the method comprises: (a) expressing SEQ ID NO:X in a cell; (b) isolating the supernatant; (c) detecting an activity in a biological assay; and (d) identifying the protein in the supernatant having the activity.
23. The product produced by the method of claim 20.

=> d his

(FILE 'HOME' ENTERED AT 11:40:44 ON 11 JUL 2002)

FILE 'BIOSIS, MEDLINE, AGRICOLA, EMBASE, CABA, WPIDS, JAPIO, BIOTECHDS, LIFESCI, CAPLUS, USPATFULL, USPAT2' ENTERED AT 11:42:50 ON 11 JUL 2002  
E LEONARD JOHN P/AU

L1 108 S E3-E5  
E LEONARD J P/AU  
L2 359 S E3-E4  
E GOLDMAN SAMUEL/AU  
L3 78 S E2-E9  
E GOLDMAN S/AU  
L4 1458 S E3  
E OHARA RICHARD/AU  
E OHARA R/AU  
L5 70 S E3  
L6 2059 S L1-L5  
L7 109 S L6 AND (IL-12 OR RA OR ARTHRITIS)  
L8 43 S L7 AND (ANTIBOD? OR ANTAGONIST?)  
L9 20 DUP REM L8 (23 DUPLICATES REMOVED)  
L10 1 S L9 AND P40

FILE 'STNGUIDE' ENTERED AT 11:51:09 ON 11 JUL 2002

L11 0 S L8 AND (FRAGMENT? OR EPITOP?)  
L12 2 S RA OR RHEUMATOID ARTHRITIS

FILE 'BIOSIS, MEDLINE, AGRICOLA, EMBASE, CABA, WPIDS, JAPIO, BIOTECHDS, LIFESCI, CAPLUS, USPATFULL, USPAT2' ENTERED AT 11:55:50 ON 11 JUL 2002

L13 3 S L9 AND (FRAGMENT? OR EPITOP?)  
L14 656173 S RA OR RHEUMATOID ARTHRITIS  
L15 1079 S L14 AND IL-12  
L16 724 S L15 AND (ANTIBOD? OR EPITOP? OR FRAGMENT? OR ANTAGONIST)  
L17 95 S L16 AND IL-12 (5A) ANTIBOD?  
L18 82 DUP REM L17 (13 DUPLICATES REMOVED)  
L19 18 S L18 AND P40  
L20 52 S L16 AND IL-12 (5A) ANTAGONIST?  
L21 44 DUP REM L20 (8 DUPLICATES REMOVED)

=> s l21 and p40

11 FILES SEARCHED...

L22 11 L21 AND P40

=> d bib 1-11

L22 ANSWER 1 OF 11 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.

AN 2001:290669 BIOSIS

DN PREV200100290669

TI Antigen-specific T cell-mediated gene therapy in collagen-induced arthritis.

AU Nakajima, Atsuo; Seroogy, Christine M.; Sandora, Matthew R.; Tarner, Ingo H.; Costa, Gina L.; Taylor-Edwards, Cariel; Bachmann, Michael H.; Contag, Christopher H.; Fathman, C. Garrison (1)

CS (1) Department of Medicine, Division of Immunology and Rheumatology, School of Medicine, Stanford University, CCSR Building, Room 2225, Stanford, CA, 94305-5111: cfathman@leland.stanford.edu USA

SO Journal of Clinical Investigation, (May, 2001) Vol. 107, No. 10, pp. 1293-1301. print.

ISSN: 0021-9738.

DT Article  
LA English  
SL English

L22 ANSWER 2 OF 11 WPIDS (C) 2002 THOMSON DERWENT  
AN 2001-244560 [25] WPIDS  
DNC C2001-073385  
TI Composition comprising interleukin-12 **p40** and IL-B30 polypeptide or its segment, useful for ameliorating **rheumatoid arthritis, osteoarthritis, atherosclerosis, multiple sclerosis, vasculitis and tumor.**  
DC B04 D16  
IN DE WAAL MALEYFT, R; KASTELEIN, R A; LIRA, S A; NARULA, S K; OPPMANN, B; RENNICK, D M; WIEKOWSKI, M T  
PA (SCHE) SCHERING CORP  
CYC 92  
PI WO 2001018051 A2 20010315 (200125)\* EN 69p  
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TZ UG ZW  
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CR CZ DE DK DM DZ EE ES FI GB GD GE HR HU ID IL IN IS JP KG KR KZ LC LK LR LT LU LV MA MD MG MK MN MX MZ NO NZ PL PT RO RU SE SG SI SK SL TJ TM TR TT TZ UA UZ VN YU ZA  
AU 2000073608 A 20010410 (200137)  
EP 1210434 A2 20020605 (200238) EN  
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT RO SE SI  
ADT WO 2001018051 A2 WO 2000-US24686 20000908; AU 2000073608 A AU 2000-73608 20000908; EP 1210434 A2 EP 2000-961688 20000908, WO 2000-US24686 20000908  
FDT AU 2000073608 A Based on WO 200118051; EP 1210434 A2 Based on WO 200118051  
PRAI US 1999-164616P 19991110; US 1999-393090 19990909  
  
L22 ANSWER 3 OF 11 BIOTECHDS COPYRIGHT 2002 THOMSON DERWENT AND ISI  
AN 2001-08257 BIOTECHDS  
TI Composition containing interleukin-12 **p40** and IL-B30 protein or its segment, useful for ameliorating **rheumatoid arthritis, osteoarthritis, atherosclerosis, multiple sclerosis, vasculitis and tumor;**  
vector-mediated gene transfer and expression in host cell,  
**antibody** and **antagonist**  
AU Oppmann B; De Waal Malefyft R; Rennick D M; Kastelein R A; Wiekowski M T; Lira S A; Narula S K  
PA Schering-USA  
LO Kenilworth, NJ, USA.  
PI WO 2001018051 15 Mar 2001  
AI WO 2000-US24686 8 Sep 2000  
PRAI US 1999-164616 10 Nov 1999; US 1999-393090 9 Sep 1999  
DT Patent  
LA English  
OS WPI: 2001-244560 [25]  
  
L22 ANSWER 4 OF 11 CAPLUS COPYRIGHT 2002 ACS  
AN 1999:487326 CAPLUS  
DN 131:129052  
TI **Antibodies against human IL-12**  
IN Gately, Maurcie Kent; Presky, David Howard  
PA F.Hoffmann-La Roche A.-G., Switz.  
SO PCT Int. Appl., 47 pp.  
CODEN: PIXXD2  
DT Patent  
LA English  
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 9937682	A2	19990729	WO 1999-EP202	19990115
	W:	AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG		
AU 9925177	A1	19990115	AU 1999-25177	19990115
CA 2318052	AA	19990729	CA 1999-2318052	19990115
BR 9907743	A	20001017	BR 1999-7743	19990115
EP 1049717	A2	20001108	EP 1999-904780	19990115
	R:	AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, PT, IE, FI		
JP 2002501085	T2	20020115	JP 2000-528602	19990115
US 6225117	B1	20010501	US 1999-232522	19990119
ZA 9900452	A	19990723	ZA 1999-452	19990121
PRAI US 1998-72333P	P	19980123		
	WO 1999-EP202	W	19990115	

L22 ANSWER 5 OF 11 USPATFULL

AN 2002:84902 USPATFULL  
 TI Nucleic acids, proteins and **antibodies**  
 IN Rosen, Craig A., Laytonsville, MD, UNITED STATES  
     Ruben, Steven M., Olney, MD, UNITED STATES  
 PI US 2002044941       A1   20020418  
 AI US 2001-925302       A1   20010810 (9)  
 RLI Continuation-in-part of Ser. No. WO 2000-US5918, filed on 8 Mar 2000,  
     UNKNOWN  
 PRAI US 1999-124270P     19990312 (60)  
 DT Utility  
 FS APPLICATION  
 LREP HUMAN GENOME SCIENCES INC, 9410 KEY WEST AVENUE, ROCKVILLE, MD, 20850  
 CLMN Number of Claims: 23  
 ECL Exemplary Claim: 1  
 DRWN No Drawings  
 LN.CNT 21121  
 CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L22 ANSWER 6 OF 11 USPATFULL

AN 2001:221075 USPATFULL  
 TI Retinoid antagonists and use thereof  
 IN Bollag, Werner, Basel, Switzerland  
     Klaus, Michael, Weil am Rhein, Germany, Federal Republic of  
     Mohr, Peter, Basel, Switzerland  
     Panina-Bordignon, Paola, Milan, Italy  
     Rosenberger, Michael, Caldwell, NJ, United States  
     Sinigaglia, Francesco, Milan, Italy  
 PA Hoffman-La Roche Inc., Nutley, NJ, United States (U.S. corporation)  
 PI US 6326397       B1   20011204  
 AI US 1999-307009     19990507 (9)  
 RLI Continuation-in-part of Ser. No. US 1998-189189, filed on 10 Nov 1998  
 DT Utility  
 FS GRANTED  
 EXNAM Primary Examiner: Killos, Paul J.  
 LREP Johnston, George W., Parise, John P.  
 CLMN Number of Claims: 16  
 ECL Exemplary Claim: 1  
 DRWN 7 Drawing Figure(s); 5 Drawing Page(s)  
 LN.CNT 1573

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L22 ANSWER 7 OF 11 USPATFULL  
AN 2001:63494 USPATFULL  
TI **Antibodies** against human IL-12  
IN Gately, Maurice Kent, Parsippany, NJ, United States  
Presky, David Howard, Glen Ridge, NJ, United States  
PA Hoffman-La Roche Inc., Nutley, NJ, United States (U.S. corporation)  
PI US 6225117 B1 20010501  
AI US 1999-232522 19990119 (9)  
PRAI US 1998-72333P 19980123 (60)  
DT Utility  
FS Granted  
EXNAM Primary Examiner: Chan, Christina Y.; Assistant Examiner: DiBrino, Marianne  
LREP Johnston, George W., Rocha-Tramaloni, Patricia S., Silverman, Robert A.  
CLMN Number of Claims: 23  
ECL Exemplary Claim: 1  
DRWN 7 Drawing Figure(s); 7 Drawing Page(s)  
LN.CNT 1122  
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L22 ANSWER 8 OF 11 USPATFULL  
AN 2000:138395 USPATFULL  
TI Treatment of T-helper cell type 2-mediated immune disease by retinoid antagonists  
IN Bollag, Werner, Basel, Switzerland  
Klaus, Michael, Weil am Rhein, Germany, Federal Republic of  
Panina-Bordignon, Paola, Milan, Italy  
Sinigaglia, Francesco, Milan, Italy  
PA Hoffmann-La Roche Inc., Nutley, NJ, United States (U.S. corporation)  
PI US 6133309 20001017  
AI US 1998-189189 19981110 (9)  
PRAI EP 1997-119776 19971112  
DT Utility  
FS Granted  
EXNAM Primary Examiner: Travers, Russell  
LREP Johnston, George W., Epstein, William H., Parise, John P.  
CLMN Number of Claims: 37  
ECL Exemplary Claim: 1  
DRWN No Drawings  
LN.CNT 780  
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L22 ANSWER 9 OF 11 USPATFULL  
AN 2000:50737 USPATFULL  
TI Methods and compositions for modulating responsiveness to corticosteroids  
IN Sekut, Les, Westborough, MA, United States  
Carter, Adam, Newburyport, MA, United States  
Ghayur, Tariq, Grafton, MA, United States  
Banerjee, Subhashis, Shrewsbury, MA, United States  
Tracey, Daniel E., Harvard, MA, United States  
PA BASF Aktiengesellschaft, Rheinland Pfalz, Germany, Federal Republic of (non-U.S. corporation)  
PI US 6054487 20000425  
AI US 1997-820692 19970318 (8)  
DT Utility  
FS Granted  
EXNAM Primary Examiner: Jarvis, William R. A.  
LREP Lahive & Cockfield, LLP  
CLMN Number of Claims: 46

ECL Exemplary Claim: 1  
DRWN 3 Drawing Figure(s); 3 Drawing Page(s)  
LN.CNT 2404  
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L22 ANSWER 10 OF 11 USPATFULL  
AN 1998:135151 USPATFULL  
TI Human receptor for interleukin-12  
IN Chua, Anne On, Wayne, NJ, United States  
Gubler, Ulrich Andreas, Glen Ridge, NJ, United States  
PA Hoffmann-La Roche Inc., Nutley, NJ, United States (U.S. corporation)  
PI US 5831007 19981103  
AI US 1995-419652 19950411 (8)  
RLI Division of Ser. No. US 1994-248532, filed on 31 May 1994, now patented,  
Pat. No. US 5536657 which is a continuation-in-part of Ser. No. US  
1993-94713, filed on 19 Jul 1993, now abandoned  
DT Utility  
FS Granted  
EXNAM Primary Examiner: Ulm, John  
LREP Johnston, George W., Epstein, William H., Bucholz, Briana C.  
CLMN Number of Claims: 10  
ECL Exemplary Claim: 1  
DRWN 35 Drawing Figure(s); 26 Drawing Page(s)  
LN.CNT 1937  
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L22 ANSWER 11 OF 11 USPATFULL  
AN 96:63048 USPATFULL  
TI Recombinant DNA encoding human receptor for interleukin-12  
IN Chua, Anne O., Wayne, NJ, United States  
Gubler, Ulrich A., Glen Ridge, NJ, United States  
PA Hoffmann-La Roche Inc., Nutley, NJ, United States (U.S. corporation)  
PI US 5536657 19960716  
AI US 1994-248532 19940531 (8)  
RLI Continuation-in-part of Ser. No. US 1993-94713, filed on 19 Jul 1993,  
now abandoned  
DT Utility  
FS Granted  
EXNAM Primary Examiner: Ulm, John  
LREP Gould, George M., Johnston, George W., Kass, Alan P.  
CLMN Number of Claims: 10  
ECL Exemplary Claim: 1  
DRWN 34 Drawing Figure(s); 25 Drawing Page(s)  
LN.CNT 1755  
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

=> d clm 8

L22 ANSWER 8 OF 11 USPATFULL  
CLM What is claimed is:  
1. A method of treating an immunoglobulin E-mediated allergic disease  
selected from the group consisting of allergic rhinitis and bronchial  
asthma, which comprises administering to a subject in need of such  
treatment having said immunoglobulin E-mediated allergic disease an  
effective amount of a retinoid **antagonist** of the formula:  
##STR7## wherein R.sup.1 is C.sub.5-10 -alkyl, or a pharmaceutically  
acceptable salt of such retinoid **antagonist** or a  
pharmaceutically acceptable hydrolyzable ester of such retinoid  
**antagonist** or its salt.  
2. The method of claim 1, wherein the administering comprises oral

administration.

3. The method of claim 2, wherein the oral administration is at a daily dosage of from about 0.05 mg to about 20 mg of the compound per kg of body weight of the subject.

4. The method of claim 3, wherein the oral administration is at a daily dosage of from about 0.3 mg to about 1.5 mg of the compound per kg of body weight of the subject.

5. The method of claim 2, wherein the oral administration comprises administering a tablet, capsule, pill or sachet containing from about 5 mg to about 200 mg of the compound.

6. The method of claim 5, wherein the oral administration comprises administering a tablet, capsule, pill or sachet containing from about 20 mg to about 100 mg of the compound.

7. The method of claim 1, wherein the administering comprises topical administration.

8. The method of claim 7, wherein the topical administration comprises administering an ointment, cream, lotion, or spray containing from about 0.01 percent to about 5.0 percent by weight of the compound.

9. The method of claim 8, wherein the topical administration comprises administering an ointment, cream, lotion, or spray containing from about 0.1 percent to about 1.0 percent by weight of the compound.

10. The method of claim 1, wherein the administering comprises inhalation.

11. The method of claim 10, wherein the inhalation comprises administering a nasal aerosol, aerosol for inhalation, or dry powder for inhalation containing from about 0.01 percent to about 5.0 percent by weight of the compound.

12. The method of claim 11, wherein the inhalation comprises administering a nasal aerosol, aerosol for inhalations, or dry powder for inhalation containing from about 0.1 percent to about 1.0 percent by weight of the compound.

13. The method of claim 1, wherein the compound is a retinoid antagonists or a alkali metal salt, alkaline earth metal salt, benzyl ester, lower alkyl ester, or 9-fluorenylmethyl ester thereof.

14. The method of claim 13, wherein the immunoglobulin E-mediated allergic disease is allergic rhinitis.

15. The method of claim 14, wherein the administering comprises oral administration.

16. The method of claim 15, wherein the oral administration is at a daily dosage of from about 0.05 mg to about 20 mg of the compound per kg of body weight of the subject.

17. The method of claim 16, wherein the oral administration is at a daily dosage of from about 0.3 mg to about 1.5 mg of the compound per kg of body weight of the subject.

18. The method of claim 15, wherein the oral administration comprises administering a tablet, capsule, pill or sachet containing from about 5

mg to about 200 mg of the compound.

19. The method of claim 18, wherein the oral administration comprises administering a tablet, capsule, pill or sachet containing from about 20 mg to about 100 mg of the compound.

20. The method of claim 19, wherein the topical administration comprises administering an ointment, cream, lotion, or spray containing from about 0.01 percent to about 5.0 percent by weight of the compound.

21. The method of claim 20, wherein the topical administration comprises administering an ointment, cream, lotion, or spray containing from about 0.1 percent to about 1.0 percent by weight of the compound.

22. The method of claim 14, the administering comprises inhalation.

23. The method of claim 22, wherein the inhalation comprises administering a nasal aerosol, aerosol for inhalation, or dry powder for inhalation containing from about 0.01 percent to about 5.0 percent by weight of the compound.

24. The method of claim 23, wherein the inhalation comprises administering a nasal aerosol, aerosol for inhalations, or dry powder for inhalation containing from about 0.1 percent to about 1.0 percent by weight of the compound.

25. The method of claim 14, wherein the compound is a retinoid antagonist or a alkali metal salt, alkaline earth metal salt, benzyl ester, lower alkyl ester, or 9-fluorenylmethyl ester thereof.

26. The method of claim 7, wherein the immunoglobulin E-mediated allergic disease is allergic bronchial asthma.

27. The method of claim 26, wherein the administering comprises oral administration.

28. The method of claim 27, wherein the oral administration is at a daily dosage of from about 0.05 mg to about 20 mg of the compound per kg of body weight of the subject.

29. The method of claim 28, wherein the oral administration is at a daily dosage of from about 0.3 mg to about 1.5 mg of the compound per kg of body weight of the subject.

30. The method of claim 27, wherein the oral administration comprises administering a tablet, capsule, pill or sachet containing from about 5 mg to about 200 mg of the compound.

31. The method of claim 30, wherein the oral administration comprises administering a tablet, capsule, pill or sachet containing from about 20 mg to about 100 mg of the compound.

32. The method of claim 31, wherein the topical administration comprises administering an ointment, cream, lotion, or spray containing from about 0.01 percent to about 5.0 percent by weight of the compound.

33. The method of claim 32, wherein the topical administration comprises administering an ointment, cream, lotion, or spray containing from about 0.1 percent to about 1.0 percent by weight of the compound.

34. The method of claim 26, wherein the administering comprises inhalation.

35. The method of claim 34, wherein the inhalation comprises administering a nasal aerosol, aerosol for inhalation, or dry powder for inhalation containing from about 0.01 percent to about 5.0 percent by weight of the compound.

36. The method of claim 35, wherein the inhalation comprises administering a nasal aerosol, aerosol for inhalations, or dry powder for inhalation containing from about 0.1 percent to about 1.0 percent by weight of the compound.

37. The method of claim 26, wherein the compound is a retinoid antagonists or a alkali metal salt, alkaline earth metal salt, benzyl ester, lower alkyl ester, or 9-fluorenylmethyl ester thereof.

=> d clm 10

L22 ANSWER 10 OF 11 USPATFULL

CLM What is claimed is:

1. A substantially pure, homogeneous and isolated low affinity human Interleukin-12 receptor protein comprising an amino acid sequence selected from SEQ ID NO:2 or SEQ ID NO:3 and which binds specifically to Interleukin-12.

2. The Interleukin-12 receptor protein of claim 1 having the amino acid sequence SEQ ID NO:2.

3. The low affinity Interleukin-12 receptor protein of claim 1 wherein the Interleukin-12 receptor protein has a K<sub>sub</sub>D of about 2 to about 10 nM.

4. The low affinity Interleukin-12 receptor protein of claim 3 wherein the Interleukin-12 receptor protein has a K<sub>sub</sub>D of about 2 to about 5 nM.

5. The low affinity Interleukin-12 receptor protein of claim 4 wherein the Interleukin-12 receptor protein has the amino acid sequence SEQ ID NO:2.

6. The low affinity Interleukin-12 receptor protein of claim 4 wherein the Interleukin-12 receptor protein has the amino acid sequence SEQ ID NO:3.

7. The Interleukin-12 receptor protein of claim 1 having the amino acid sequence SEQ ID NO:3.

8. A pharmaceutical composition comprising a substantially pure, homogeneous and isolated low affinity human Interleukin-12 receptor protein comprising an amino acid sequence selected from SEQ ID NO:2 or SEQ ID NO:3 and which binds specifically to Interleukin-12 and a suitable diluent or carrier.

9. The pharmaceutical composition of claim 8 wherein the human low affinity Interleukin-12 receptor protein has the amino acid sequence SEQ ID NO:2.

10. The pharmaceutical composition of claim 8 wherein the human low affinity Interleukin-12 receptor protein has the amino acid sequence SEQ ID NO:3.

=> d his

(FILE 'HOME' ENTERED AT 11:40:44 ON 11 JUL 2002)

FILE 'BIOSIS, MEDLINE, AGRICOLA, EMBASE, CABA, WPIDS, JAPIO, BIOTECHDS,  
LIFESCI, CAPLUS, USPATFULL, USPAT2' ENTERED AT 11:42:50 ON 11 JUL 2002

E LEONARD JOHN P/AU

L1 108 S E3-E5

E LEONARD J P/AU

L2 359 S E3-E4

E GOLDMAN SAMUEL/AU

L3 78 S E2-E9

E GOLDMAN S/AU

L4 1458 S E3

E OHARA RICHARD/AU

E OHARA R/AU

L5 70 S E3

L6 2059 S L1-L5

109 S L6 AND (IL-12 OR RA OR ARTHRITIS)

L8 43 S L7 AND (ANTIBOD? OR ANTAGONIST?)

L9 20 DUP REM L8 (23 DUPLICATES REMOVED)

L10 1 S L9 AND P40

FILE 'STNGUIDE' ENTERED AT 11:51:09 ON 11 JUL 2002

L11 0 S L8 AND (FRAGMENT? OR EPITOP?)

L12 2 S RA OR RHEUMATOID ARTHRITIS

FILE 'BIOSIS, MEDLINE, AGRICOLA, EMBASE, CABA, WPIDS, JAPIO, BIOTECHDS,  
LIFESCI, CAPLUS, USPATFULL, USPAT2' ENTERED AT 11:55:50 ON 11 JUL 2002

3 S L9 AND (FRAGMENT? OR EPITOP?)

L14 656173 S RA OR RHEUMATOID ARTHRITIS

L15 1079 S L14 AND IL-12

724 S L15 AND (ANTIBOD? OR EPITOP? OR FRAGMENT? OR ANTAGONIST?)

L17 95 S L16 AND IL-12 (5A) ANTIBOD?

L18 82 DUP REM L17 (13 DUPLICATES REMOVED)

L19 18 S L18 AND P40

L20 52 S L16 AND IL-12 (5A) ANTAGONIST?

L21 44 DUP REM L20 (8 DUPLICATES REMOVED)

L22 11 S L21 AND P40

=> s l16 and p40

L23 67 L16 AND P40

=> s l23 and (antibod? or antagonist?)

L24 67 L23 AND (ANTIBOD? OR ANTAGONIST?)

=> dup rem l24

PROCESSING COMPLETED FOR L24

L25 55 DUP REM L24 (12 DUPLICATES REMOVED)

=> s l25 and (antibod? (5a) IL-12 or IL-12 (5a) antagonist?)

8 FILES SEARCHED...

L26 21 L25 AND (ANTIBOD? (5A) IL-12 OR IL-12 (5A) ANTAGONIST?)

=> d bib ab 1-21

L26 ANSWER 1 OF 21 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.

AN 2001:290669 BIOSIS

DN PREV200100290669

TI Antigen-specific T cell-mediated gene therapy in collagen-induced

arthritis.

AU Nakajima, Atsuo; Seroogy, Christine M.; Sandora, Matthew R.; Turner, Ingo H.; Costa, Gina L.; Taylor-Edwards, Cariel; Bachmann, Michael H.; Contag, Christopher H.; Fathman, C. Garrison (1)

CS (1) Department of Medicine, Division of Immunology and Rheumatology, School of Medicine, Stanford University, CCSR Building, Room 2225, Stanford, CA, 94305-5111: cfathman@leland.stanford.edu USA

SO Journal of Clinical Investigation, (May, 2001) Vol. 107, No. 10, pp. 1293-1301. print.  
ISSN: 0021-9738.

DT Article

LA English

SL English

AB Autoantigen-specific T cells have tissue-specific homing properties, suggesting that these cells may be ideal vehicles for the local delivery of immunoregulatory molecules. We tested this hypothesis by using type II collagen-specific (CII-specific) CD4+ T hybridomas or primary CD4+ T cells after gene transfer, as vehicles to deliver an immunoregulatory protein for the treatment of collagen-induced arthritis (CIA), a mouse model of **rheumatoid arthritis (RA)**. CII-specific T cells or hybridomas were transduced using retroviral vectors to constitutively express the **IL-12 antagonist, IL-12 p40**. Transfer of engineered CD4+ T cells after immunization significantly inhibited the development of CIA, while cells transduced with vector control had no effect. The beneficial effect on CIA of **IL-12 p40**-transduced T cells required TCR specificity against CII, since transfer of T cells specific for another antigen producing equivalent amounts of **IL-12 p40** had no effect. In vivo cell detection using bioluminescent labels and RT-PCR showed that transferred CII-reactive T-cell hybridomas accumulated in inflamed joints in mice with CIA. These results indicate that the local delivery of **IL-12 p40** by T cells inhibited CIA by suppressing autoimmune responses at the site of inflammation. Modifying antigen-specific T cells by retroviral transduction for local expression of immunoregulatory proteins thus offers a promising strategy for treating **RA**.

L26 ANSWER 2 OF 21 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.

AN 1999:518318 BIOSIS

DN PREV199900518318

TI Differential regulation of rheumatoid synovial cell interleukin-12 production by tumor necrosis factor alpha and CD40 signals.

AU Kitagawa, Minetake (1); Mitsui, Hiroshi; Nakamura, Hiroshi; Yoshino, Shinichi; Miyakawa, Shunpei; Ochiai, Naoyuki; Onobori, Makoto; Suzuki, Hiroshi; Sumida, Takayuki

CS (1) Division of Rheumatology, Department of Internal Medicine, Institute of Clinical Medicine, University of Tsukuba, 1-1-1 Tennodai, Tsukuba-shi, Ibaraki, 305-8575 Japan

SO Arthritis & Rheumatism, (Sept., 1999) Vol. 42, No. 9, pp. 1917-1926.  
ISSN: 0004-3591.

DT Article

LA English

SL English

AB Objective: To investigate the roles of tumor necrosis factor alpha (TNF $\alpha$ ) and the CD40-CD154 interaction in interleukin-12 (**IL-12**) production by rheumatoid synovial cells (SC). Methods: Levels of **IL-12 (p40 and p70)** in synovial tissue and culture supernatants of SC from patients with **rheumatoid arthritis (RA)**, osteoarthritis (OA), and ankylosing spondylitis (AS) were assayed by enzyme-linked immunosorbent assay. Effects of anti-CD154 and anti-TNF $\alpha$  **antibody** on spontaneous and lipopolysaccharide (LPS)-stimulated **IL-12**

production by SC were examined. Effects of immobilized anti-CD3 treatment and depletion of CD4+ T cells on **IL-12** production were also tested. CD154 expression by synovial T cells and intracellular **IL-12** production during culture were analyzed by flow cytometry. Results: **IL-12 p40** and p70 levels in **RA** synovial tissue and spontaneous **IL-12** **p40** production by SC from **RA** patients were significantly higher than the levels in OA and AS patients. Spontaneous **IL-12** production by SC from **RA** patients significantly decreased after depletion of CD4+ T cells from SC or after application of anti-CD154 **antibody**, but not by treatment with anti-TNFalpha **antibody**. Anti-CD3 **antibody** stimulation increased spontaneous **IL-12 p40** production and CD154 expression by synovial T cells. The increment of **IL-12 p40** production by anti-CD3 was abrogated by anti-CD154 **antibody**. **IL-12 p40** production was also increased by LPS stimulation. LPS-stimulated **IL-12** production was inhibited by anti-TNFalpha **antibody**, but not by T cell depletion and anti-CD154 **antibody** treatment. The TNFalpha inhibitor rolipram inhibited LPS-stimulated **IL-12 p40** production by **RA** SC more strongly than spontaneous production. TNFalpha restored LPS-stimulated **IL-12** production that had been inhibited by rolipram. Conclusion: **IL-12** production in **RA** is regulated by 2 different pathways. One pathway is T cell dependent, predominantly through a CD40-CD154 interaction, while the other is T cell independent, mediated through TNFalpha. Inhibition of **IL-12** production by interference with CD40-CD154 interaction and TNFalpha production may be a potential therapeutic strategy for treating **RA**.

L26 ANSWER 3 OF 21 WPIDS (C) 2002 THOMSON DERWENT  
AN 2001-244560 [25] WPIDS  
DNC C2001-073385  
TI Composition comprising interleukin-12 **p40** and IL-B30 polypeptide or its segment, useful for ameliorating **rheumatoid arthritis**, osteoarthritis, atherosclerosis, multiple sclerosis, vasculitis and tumor.  
DC B04 D16  
IN DE WAAL MALEYFT, R; KASTELEIN, R A; LIRA, S A; NARULA, S K; OPPMANN, B; RENNICK, D M; WIEKOWSKI, M T  
PA (SCHE) SCHERING CORP  
CYC 92  
PI WO 2001018051 A2 20010315 (200125)\* EN 69p  
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ  
NL OA PT SD SE SL SZ TZ UG ZW  
W: AE AG AL AM AT AU AZ BA BB BG BR BY BZ CA CH CN CR CZ DE DK DM DZ  
EE ES FI GB GD GE HR HU ID IL IN IS JP KG KR KZ LC LK LR LT LU LV  
MA MD MG MK MN MX MZ NO NZ PL PT RO RU SE SG SI SK SL TJ TM TR TT  
TZ UA UZ VN YU ZA  
AU 2000073608 A 20010410 (200137)  
EP 1210434 A2 20020605 (200238) EN  
R: AL AT BE CH CY DE DK ES FI FR GB GR IE IT LI LT LU LV MC MK NL PT  
RO SE SI  
ADT WO 2001018051 A2 WO 2000-US24686 20000908; AU 2000073608 A AU 2000-73608  
20000908; EP 1210434 A2 EP 2000-961688 20000908, WO 2000-US24686 20000908  
FDT AU 2000073608 A Based on WO 200118051; EP 1210434 A2 Based on WO 200118051  
PRAI US 1999-164616P 19991110; US 1999-393090 19990909  
AB WO 200118051 A UPAB: 20010508  
NOVELTY - A composition (I) comprising a substantially pure polypeptide comprising a number of distinct segments of at least 7 contiguous amino acids from interleukin (**IL**)-**12 p40** and/or **IL-B30**, and a substantially pure polypeptide comprising a segment of at

least 11 contiguous amino acids from **IL-12 p40**  
and/or **IL-B30**.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

- (1) an isolated or recombinant nucleic acid (II) encoding (I);
- (2) a cell (III) comprising (II);
- (3) a nucleic acid (IV) which hybridizes under wash conditions of 30 minutes at 50 deg. C and less than 1M salt to the natural mature coding portion of primate **IL-12 p40** and **IL-B30**;
- (4) an **antagonist** (V) of **IL-12 p40/IL-B30** combined with a tumor necrosis factor-alpha (TNF alpha ) **antagonist**, an **IL-12 antagonist**, **IL-10**, or steroids;
- (5) a binding compound (VI) comprising an antigen binding site from an **antibody**, which specifically binds to (I) and comprising a substantially pure polypeptide comprising **IL-12 p40** and **IL-B30** polypeptide, or a polypeptide comprising **IL-12 p40** fused to **IL-B30**, but not to either **IL-12 p40** or **IL-B30** polypeptide;
- (6) a kit (VII) comprising:
  - (a) (I), and a compartment comprising the polypeptide, or instructions for use or disposal of reagents in the kit;
  - (b) (II), and a compartment comprising (II), a compartment further comprising a primate **IL-12 p40** or **IL-B30**, or instructions for use or disposal of reagents in the kit or (VI); and
  - (c) a compartment comprising (VI), or instructions for use or disposal of reagents in the kit;
- (7) producing (M1) an antigen:**antibody** complex, involves contacting, under appropriate conditions, a primate **IL-12 p40/IL-B30** composition with (VI), allowing the complex to form;
- (8) a composition (VIII) comprising (VI) which is sterile, or (VI) and a carrier such as an aqueous compound, including water, saline, and/or buffer;
- (9) increasing (M2) the secretion of a primate **IL-B30**, involves expressing the polypeptide with **IL-12 p40** or increasing the secretion of a primate **IL-12 p40** involves expressing the **IL-12 p40** with **IL-B30**; and
- (10) screening (M3) for a receptor which binds (I) involves contacting the complex to a cell expressing the receptor under conditions allowing the complex to bind to the receptor, forming a detectable interaction.

ACTIVITY - Antirheumatic; antiarthritic; osteopathic; antiarthritic; neuroprotective; antiarteriosclerotic; cerebroprotective; vasotropic; cytostatic; antitumor; immunosuppressive.

MECHANISM OF ACTION - Modulator of physiology or development of cell in host; inducer of memory T-cell proliferation (claimed); modulator of trafficking or activation of leukocyte.

No supporting data is given.

USE - (I) is useful for modulating physiology or development of a cell or tissue in a host organism by contacting the cell with (I) or (V), resulting in an increased or decreased production of Interferon-gamma (IFN gamma ), an enhanced Th1 response such as anti-tumor effect, adjuvant effect, anti-viral effect or antagonized allergic effect, and amelioration of an autoimmune condition or a chronic inflammatory condition. The contacting is in combination with **IL-18**, **IL-12**, radiation therapy or chemotherapy, an immune adjuvant or an anti-viral therapeutic. The **antagonist** is an **antibody** against **IL-12** receptor subunit beta 1. The **antagonist** or agonist of mammalian **IL-B30** protein is useful for modulating the inflammatory response in an animal, by contacting cells in the animal with

the agonist or **antagonist**, where the animal exhibits signs or symptoms of an acute phase inflammatory response in skin, lung, gastrointestinal, or liver tissue. The modulation is accelerating maturation of neutrophils into platelets and has an effect on immunoglobulin A and G (IgA and IgG) . The **antagonist** is an **antibody** which binds to the mammalian IL-B30 or blocks signaling mediated by mammalian IL-B30. The **antagonist** or agonist is administered in combination with an anti-inflammatory cytokine agonist or **antagonist**, an analgesic, an anti-inflammatory agent, or a steroid. IL-B30 or its agonist is useful inducing the proliferation of memory T-cells (all claimed).

Agonist or **antagonist** of IL-B30 protein is useful for modulating the trafficking or activation of a leukocyte in an animal experiencing science or symptoms of autoimmunity, an inflammatory condition, tissue specific autoimmunity, degenerative autoimmunity, **rheumatoid arthritis**, osteoarthritis, atherosclerosis, multiple sclerosis, vasculitis, delayed hypersensitivities, skin grafting, a transplant, spinal injury, stroke, neurodegeneration, an infectious disease, ischemia, cancer, tumors, multiple myeloma, Castleman's disease, postmenopausal osteoporosis or IL-6-associated diseases.

**IL-12 p40/IL-B30** is useful as an immunogen for the production a antisera or **antibodies** specific for binding. (I) is useful for in vitro assays, scientific research, and the synthesis or manufacture of nucleic acids or **antibodies**. (II) is useful in forensic science.

Dwg.0/0

L26 ANSWER 4 OF 21 WPIDS (C) 2002 THOMSON DERWENT  
AN 1999-458684 [38] WPIDS  
DNC C1999-134705  
TI New **antibodies** to human interleukin-12, used for treating diseases associated with increased **IL-12** bioactivity such as autoimmune disorders, e.g. multiple sclerosis.  
DC B04 D16  
IN GATELY, M K; PRESKY, D H; GATELY, M  
PA (HOFF) HOFFMANN LA ROCHE & CO AG F; (HOFF) HOFFMANN LA ROCHE INC  
CYC 85  
PI WO 9937682 A2 19990729 (199938)\* EN 46p  
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL  
OA PT SD SE SZ UG ZW  
W: AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GE  
GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD  
MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT UA  
UG UZ VN YU ZW  
ZA 9900452 A 19990929 (199947) 48p  
AU 9925177 A 19990809 (200001)  
BR 9907743 A 20001017 (200056)  
EP 1049717 A2 20001108 (200062) EN  
R: AT BE CH CY DE DK ES FI FR GB GR IE IT LI LU NL PT SE  
US 6225117 B1 20010501 (200126)  
CN 1288468 A 20010321 (200137)  
KR 2001034315 A 20010425 (200164)  
MX 2000007124 A1 20010301 (200170)  
JP 2002501085 W 20020115 (200207) 50p  
ADT WO 9937682 A2 WO 1999-EP202 19990115; ZA 9900452 A ZA 1999-452 19990121;  
AU 9925177 A AU 1999-25177 19990115; BR 9907743 A BR 1999-7743 19990115,  
WO 1999-EP202 19990115; EP 1049717 A2 EP 1999-904780 19990115, WO  
1999-EP202 19990115; US 6225117 B1 Provisional US 1998-72333P 19980123, US  
1999-232522 19990119; CN 1288468 A CN 1999-802310 19990115; KR 2001034315  
A KR 2000-708036 20000722; MX 2000007124 A1 MX 2000-7124 20000720; JP  
2002501085 W WO 1999-EP202 19990115, JP 2000-528602 19990115  
FDT AU 9925177 A Based on WO 9937682; BR 9907743 A Based on WO 9937682; EP

1049717 A2 Based on WO 9937682; JP 2002501085 W Based on WO 9937682  
PRAI US 1998-72333P 19980123; US 1999-232522 19990119  
AB WO 9937682 A UPAB: 19991122

NOVELTY - New **antibodies** to human interleukin-12 are produced using a mammal which is deficient in the gene encoding the p35 or **p40** subunit of **IL-12**.

DETAILED DESCRIPTION - (A) An **antibody** to the human interleukin (**IL**)-12 p75 heterodimer which consists of a p35 subunit and a **p40** subunit, where the **antibody**:

(i) immunologically reacts with an **epitope** presented by the p75 heterodimer of human **IL-12**, but is not immunologically reactive with an **epitope** presented by the **p40** subunit; and

(ii) is produced from a mammal, preferably a mouse which is deficient in the gene encoding the p35 subunit or the **p40** subunit of **IL-12**.

INDEPENDENT CLAIMS are also included for the following:

(1) a monoclonal **antibody** (MAb) to human **IL-12** which consists of a p35 subunit and a **p40** subunit forming a p75 heterodimer, where the MAb;

(i) immunologically reacts with an **epitope** presented by the p75 heterodimer of human **IL-12**, but is not immunologically reactive with any **epitope** presented by the **p40** subunit; and

(ii) neutralizes at least 90% of the bioactivity of human **IL-12**;

(2) a hybridoma that produces an **antibody** as in (A) or (1).

ACTIVITY - The **antibodies** can neutralize **IL-12** bioactivity as determined by ability to block **IL-12** stimulated phytohemagglutinin A (PHA)-activated lymphoblast proliferation and interferon-gamma production by PHA-activated lymphoblasts. The 5F2, 16F2, 16G2 and 20E11 **antibodies** were able to inhibit human **IL-12** stimulated PHA activated human lymphoblast proliferation by at least 90%. These anti-human heterodimer specific **IL-12 antibodies** were able to inhibit greater than 90% of **IL-12** stimulated IFN-gamma production when used at 0.5 micro g/ml.

USE - The **antibodies** can be used for controlling diseases with pathologies that are mediated through immune mechanisms, particularly diseases associated with increased **IL-12** bioactivity that results in aberrant Th1-type helper cell activity like autoimmune disorders, e.g. multiple sclerosis, **rheumatoid arthritis**, autoimmune diabetes mellitus, and inflammatory bowel disease (IBD) including Crohn's disease and ulcerative colitis (claimed). They can also be used to treat transplantation/graff-versus-host disease and septic shock.

ADVANTAGE - The anti-**IL-12 antibodies** exhibit higher potency and greater efficacy than known heterodimer specific **IL-12 antibodies**.

Dwg.0/7

L26 ANSWER 5 OF 21 BIOTECHDS COPYRIGHT 2002 THOMSON DERWENT AND ISI  
AN 2001-08257 BIOTECHDS

TI Composition containing interleukin-12 **p40** and IL-B30 protein or its segment, useful for ameliorating **rheumatoid arthritis**, osteoarthritis, atherosclerosis, multiple sclerosis, vasculitis and tumor;

vector-mediated gene transfer and expression in host cell,  
**antibody** and **antagonist**

AU Oppmann B; De Waal Malefyt R; Rennick D M; Kastelein R A; Wiekowski M T;  
Lira S A; Narula S K

PA Schering-USA

LO Kenilworth, NJ, USA.  
PI WO 2001018051 15 Mar 2001  
AI WO 2000-US24686 8 Sep 2000  
PRAI US 1999-164616 10 Nov 1999; US 1999-393090 9 Sep 1999  
DT Patent  
LA English  
OS WPI: 2001-244560 [25]  
AB A composition containing a substantially pure protein containing a number of distinct segments of at least 7 contiguous amino acids from interleukin (IL)-12 p40 and/or IL-B30, and a substantially pure protein containing a segment of at least 11 contiguous amino acids from IL-12 p40 and/or IL-B30, is new. Also claimed are: a recombinant nucleic acid encoding the protein; a cell containing the nucleic acid; a nucleic acid which hybridizes under wash conditions of 30 min at 50 deg and less than 1M salt to the natural mature coding portion of primate IL-12 p40 and IL-B30; an **antagonist** of IL-12 p40/IL-B30 combined with a tumor necrosis factor-alpha (TNF-alpha) **antagonist**, an IL-12 **antagonist**, IL-10 or steroids; a binding compound containing an antigen binding site from an **antibody** which specifically binds to the protein; a kit containing the composition, polynucleotide and a binding compound; producing an antigen: **antibody** complex; a composition containing a binding compound; increasing the secretion of a primate IL-B30; and screening for a receptor which binds the composition. The composition is useful for modulating physiology or development of a cell or tissue0. (69pp)

L26 ANSWER 6 OF 21 USPATFULL  
AN 2002:157653 USPATFULL  
TI Triazine compounds  
IN Ono, M, Lexington, MA, UNITED STATES  
Sun, Lijun, Harvard, MA, UNITED STATES  
Zhang, Shijie, Nashua, NH, UNITED STATES  
Przewloka, Teresa, Burlington, MA, UNITED STATES  
James, David A., Cambridge, MA, UNITED STATES  
Ding, Wenli, Worcester, MA, UNITED STATES  
Wada, Yumiko, Waltham, MA, UNITED STATES  
PI US 2002082259 A1 20020627  
AI US 2001-6624 A1 20011130 (10)  
RLI Continuation-in-part of Ser. No. US 2000-594362, filed on 15 Jun 2000,  
PENDING  
DT Utility  
FS APPLICATION  
LREP Y. ROCKY TSAO, Fish & Richardson P.C., 225 Franklin Street, Boston, MA,  
02110-2804  
CLMN Number of Claims: 48  
ECL Exemplary Claim: 1  
DRWN No Drawings  
LN.CNT 879  
AB This invention relates to triazine compounds of formula (I): ##STR1##

R.sub.1 is , aryl, ##STR2##

or heteroaryl; each of R.sub.2, R.sub.4, and R.sub.5, independently, is R.sup.c, halogen, nitro, nitroso, cyano, azide, isothionitro, SR.sup.c, or OR.sup.c; R.sub.3 is R.sup.c, alkenyl, alkynyl, aryl, heteroaryl, cyclyl, heterocyclyl, OR.sup.c, OC(O)R.sup.c, SO.sub.2R.sup.c, S(O)R.sup.c, S(O.sub.2)NR.sup.cR.sup.d, SR.sup.c, NR.sup.cR.sup.d, NR.sup.cCOR.sup.d, NR.sup.cC(O)OR.sup.d, NR.sup.cC(O)NR.sup.cR.sup.d, NR.sup.cSO.sub.2R.sup.d, COR.sup.c, C(O)OR.sup.c, or C(O)NR.sup.cR.sup.d; n is 0, 1, 2, 3, 4, 5, 6, or 7; X is O, S, S(O),

S(O.<sub>sub.</sub>2), or NR.sup.c; Y is a covalent bond, CH.<sub>sub.</sub>2, C(O), C.dbd.N--R.sup.c, C.dbd.N--OR.sup.c, C.dbd.N--SR.sup.c, O, S, S(O), or S(O.<sub>sub.</sub>2); Z is N; and W is O, S, S(O), S(O.<sub>sub.</sub>2), NR.sup.c, or NC(O)R.sup.c; in which each of R.sup.a and R.sup.b, independently, is H, alkyl, aryl, heteroaryl; and each of R.sup.c and R.sup.d, independently, is H, alkyl, or alkylcarbonyl.

L26 ANSWER 7 OF 21 USPATFULL  
AN 2002:84902 USPATFULL  
TI Nucleic acids, proteins and **antibodies**  
IN Rosen, Craig A., Laytonsville, MD, UNITED STATES  
Ruben, Steven M., Olney, MD, UNITED STATES  
PI US 2002044941 A1 20020418  
AI US 2001-925302 A1 20010810 (9)  
RLI Continuation-in-part of Ser. No. WO 2000-US5918, filed on 8 Mar 2000,  
UNKNOWN  
PRAI US 1999-124270P 19990312 (60)  
DT Utility  
FS APPLICATION  
LREP HUMAN GENOME SCIENCES INC, 9410 KEY WEST AVENUE, ROCKVILLE, MD, 20850  
CLMN Number of Claims: 23  
ECL Exemplary Claim: 1  
DRWN No Drawings  
LN.CNT 21121

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention relates to novel lung cancer related polynucleotides, the polypeptides encoded by these polynucleotides herein collectively referred to as "lung cancer antigens," and **antibodies** that immunospecifically bind these polypeptides, and the use of such lung cancer polynucleotides, antigens, and **antibodies** for detecting, treating, preventing and/or prognosing disorders of the lung, including, but not limited to, the presence of lung cancer and lung cancer metastases. More specifically, isolated lung cancer nucleic acid molecules are provided encoding novel lung cancer polypeptides. Novel lung cancer polypeptides and **antibodies** that bind to these polypeptides are provided. Also provided are vectors, host cells, and recombinant and synthetic methods for producing human lung cancer polynucleotides, polypeptides, and/or **antibodies**. The invention further relates to diagnostic and therapeutic methods useful for diagnosing, treating, preventing and/or prognosing disorders related to the lung, including lung cancer, and therapeutic methods for treating such disorders. The invention further relates to screening methods for identifying agonists and **antagonists** of polynucleotides and polypeptides of the invention. The invention further relates to methods and/or compositions for inhibiting or promoting the production and/or function of the polypeptides of the invention.

L26 ANSWER 8 OF 21 USPATFULL  
AN 2001:229210 USPATFULL  
TI Methods for enhancing oral tolerance and treating autoimmune disease using inhibitors of interleukin-12  
IN Strober, Warren, Bethesda, MD, United States  
Kelsall, Brian, Washington, DC, United States  
Marth, Thomas, Kensington, MD, United States  
PA Government of the United States of America, Department of Health and Human Services (U.S. corporation)  
PI US 2001051159 A1 20011213  
AI US 2000-732502 A1 20001207 (9)  
RLI Continuation of Ser. No. US 1999-284169, filed on 9 Apr 1999, ABANDONED  
A 371 of International Ser. No. WO 1996-US16007, filed on 11 Oct 1996,  
UNKNOWN  
DT Utility

FS APPLICATION  
LREP mary l. miller THE CANDLER BUILDING, needle & rosenberg, p.c., 127  
peachtree street, n.e., atlanta, GA, 30303-1811  
CLMN Number of Claims: 20  
ECL Exemplary Claim: 1  
DRWN No Drawings  
LN.CNT 1252  
CAS INDEXING IS AVAILABLE FOR THIS PATENT.  
AB The present invention provides a method for enhancing oral tolerance to an antigen associated with an autoimmune disease in a subject having the autoimmune disease comprising orally administering to the subject an antigen associated with the autoimmune disease and administering an inhibitor of interleukin-12 in amounts sufficient to enhance oral tolerance. Also provided in the present invention is a method for treating or preventing an autoimmune disease in a subject comprising orally administering to the subject an antigen associated with the autoimmune disease and administering an inhibitor of interleukin-12 in amounts sufficient to treat or prevent the autoimmune disease, thereby treating or preventing the autoimmune disease.

L26 ANSWER 9 OF 21 USPATFULL  
AN 2001:221075 USPATFULL  
TI Retinoid **antagonists** and use thereof  
IN Bollag, Werner, Basel, Switzerland  
Klaus, Michael, Weil am Rhein, Germany, Federal Republic of  
Mohr, Peter, Basel, Switzerland  
Panina-Bordignon, Paola, Milan, Italy  
Rosenberger, Michael, Caldwell, NJ, United States  
Sinigaglia, Francesco, Milan, Italy  
PA Hoffman-La Roche Inc., Nutley, NJ, United States (U.S. corporation)  
PI US 6326397 B1 20011204  
AI US 1999-307009 . 19990507 (9)  
RLI Continuation-in-part of Ser. No. US 1998-189189, filed on 10 Nov 1998  
DT Utility  
FS GRANTED  
EXNAM Primary Examiner: Killos, Paul J.  
LREP Johnston, George W., Parise, John P.  
CLMN Number of Claims: 16  
ECL Exemplary Claim: 1  
DRWN 7 Drawing Figure(s); 5 Drawing Page(s)  
LN.CNT 1573  
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention relates to novel retinoid **antagonists** of the formula I ##STR1##

wherein the dotted bond can be either hydrogenated or form a double bond; and, when the dotted bond forms a double bond, R.<sup>1</sup> is lower alkyl and R.<sup>2</sup> is hydrogen; and, when the dotted bond is hydrogenated, R.<sup>1</sup> and R.<sup>2</sup> taken together are methylene to form a cis-substituted cyclopropyl ring; R.<sup>3</sup> is hydroxy or lower alkoxy; R.<sup>4</sup> is alkyl or alkoxy; and R.<sup>5</sup> and R.<sup>6</sup> are, independently, a C<sub>4-12</sub> alkyl or a 5-12 cycloalkyl substituent containing from 1-3 rings which are either unsubstituted or substituted with from 1-3 lower alkyl groups, with the carbon atom of R.<sup>5</sup> and R.<sup>6</sup> being linked to the remainder of the molecule to form a quaternary carbon atom pharmaceutically acceptable salts of carbocyclic acids of the formula I; as well as method for the treatment of osteoporosis and preneoplastic and neoplastic diseases, and a method for reducing or abolishing adverse events in subjects receiving retinoid agonist treatment by administering a retinoid **antagonist**.

L26 ANSWER 10 OF 21 USPATFULL

AN 2001:63494 USPATFULL  
TI **Antibodies** against human **IL-12**  
IN Gately, Maurice Kent, Parsippany, NJ, United States  
Presky, David Howard, Glen Ridge, NJ, United States  
PA Hoffman-La Roche Inc., Nutley, NJ, United States (U.S. corporation)  
PI US 6225117 B1 20010501  
AI US 1999-232522 19990119 (9)  
PRAI US 1998-72333P 19980123 (60)  
DT Utility  
FS Granted  
EXNAM Primary Examiner: Chan, Christina Y.; Assistant Examiner: DiBrino, Marianne  
LREP Johnston, George W., Rocha-Tramaloni, Patricia S., Silverman, Robert A.  
CLMN Number of Claims: 23  
ECL Exemplary Claim: 1  
DRWN 7 Drawing Figure(s); 7 Drawing Page(s)  
LN.CNT 1122

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The present invention relates to novel p75 heterodimer specific anti-human **IL-12 antibodies** that are characterized by a higher potency and greater efficacy in neutralizing human **IL-12** bioactivity than known heterodimer specific **IL-12 monoclonal antibodies**. The heterodimer specific **antibodies** recognize one or more **epitopes** of the human **IL-12** p75 heterodimer, but do not bind to the **p40** subunit alone. The heterodimer specific **IL-12 antibodies** neutralize rhesus monkey **IL-12** bioactivity with a potency similar to their potency for neutralizing human **IL-12** bioactivity making them useful **IL-12 antagonists** for *in vivo* studies in the rhesus monkey.

L26 ANSWER 11 OF 21 USPATFULL  
AN 2000:138395 USPATFULL  
TI Treatment of T-helper cell type 2-mediated immune disease by retinoid **antagonists**  
IN Bollag, Werner, Basel, Switzerland  
Klaus, Michael, Weil am Rhein, Germany, Federal Republic of  
Panina-Bordignon, Paola, Milan, Italy  
Sinigaglia, Francesco, Milan, Italy  
PA Hoffmann-La Roche Inc., Nutley, NJ, United States (U.S. corporation)  
PI US 6133309 20001017  
AI US 1998-189189 19981110 (9)  
PRAI EP 1997-119776 19971112  
DT Utility  
FS Granted  
EXNAM Primary Examiner: Travers, Russell  
LREP Johnston, George W., Epstein, William H., Parise, John P.  
CLMN Number of Claims: 37  
ECL Exemplary Claim: 1  
DRWN No Drawings  
LN.CNT 780

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Retinoids with retinoid receptor **antagonistic** activity, pharmaceutically acceptable salts and pharmaceutically acceptable hydrolyzable esters thereof, have been found efficacious in treating T-helper cell type 2 (Th2)-mediated immune diseases, such as immunoglobulin E (IgE)-mediated allergic diseases.

L26 ANSWER 12 OF 21 USPATFULL  
AN 2000:98551 USPATFULL  
TI Treatment of papillomavirus-associated lesions

IN Stanley, Margaret Anne, Cambridge, United Kingdom  
Scarpini, Cinzia Giuseppina, Cambridge, United Kingdom  
PA Cambridge University Technical Services, Ltd., Cambridge, United Kingdom  
(non-U.S. corporation)  
PI US 6096869 20000801  
AI US 1996-621841 19960322 (8)  
PRAI GB 1995-5784 19950322  
DT Utility  
FS Granted  
EXNAM Primary Examiner: Park, Hankyel  
LREP Klarquist Sparkman Campbell Leigh & Winston, LLP  
CLMN Number of Claims: 13  
ECL Exemplary Claim: 1  
DRWN No Drawings  
LN.CNT 1293

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Interleukin-12 (**IL-12**) or a functional analogue thereof, or a polynucleotide encoding **IL-12** or encoding a functional analogue thereof, is used as a therapeutic material or adjuvant in treating papillomavirus-associated lesions e.g. warts due to HPV 6 and/or 11, e.g. condyloma acuminata. **IL-12** or a vector encoding it for endogenous production can be used together with a vaccine such as a papillomavirus antigen, or a vector encoding a papillomavirus antigen.

L26 ANSWER 13 OF 21 USPATFULL  
AN 2000:87729 USPATFULL  
TI Method of converting a Th2-type allergic immune response into a Th1-type immune response  
IN DeKruyff, Rosemarie H., Stanford, CA, United States  
Umetsu, Dale T., Stanford, CA, United States  
PA The Board of Trustees of the Leland Stanford Junior University, Palo Alto, CA, United States (U.S. corporation)  
PI US 6086898 20000711  
AI US 1999-339068 19990623 (9)  
PRAI US 1998-90390P 19980623 (60)  
DT Utility  
FS Granted  
EXNAM Primary Examiner: Chan, Christina Y.; Assistant Examiner: Ewoldt, Gerald R.  
LREP Bozicevic, Field & Francis, Sherwood, Pamela  
CLMN Number of Claims: 19  
ECL Exemplary Claim: 1  
DRWN 17 Drawing Figure(s); 10 Drawing Page(s)  
LN.CNT 1767

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Methods are provided for the treatment of allergic and other immune disorders associated with overproduction of Th2 type cytokines by antigen specific T cells. Immunotherapy with adjuvants, as provided in the present invention, greatly inhibits the development of airway hyperreactivity and airway inflammation. Such immunotherapy is shown to reverse ongoing airway disease, and convert allergic inflammatory responses into protective immune responses. Conditions of particular interest include allergic conditions associated with production of Th2 cytokines and/or IgE **antibodies**, asthma, allergic rhinitis, and anaphylactic reactions. The addition of adjuvant induces a Th1-type immune response and can redirect an established Th2-type response to a Th1-type response for the selected antigen. Preferably, antigen-specific IgE production is reduced without altering the intensity of the antigen-specific proliferative response. One particularly preferred adjuvant for use in accordance with the present invention is a Listeria adjuvant.

L26 ANSWER 14 OF 21 USPATFULL  
AN 2000:87707 USPATFULL  
TI Methods and compositions for the inhibition of interleukin-12 production  
IN Karp, Christopher L., Lutherville, MD, United States  
Trinchieri, Giorgio, Wynnewood, PA, United States  
Wysocka, Maria, Wynnewood, PA, United States  
Griffin, Diane E., Hunt Valley, MD, United States  
PA The Wistar Institute, Philadelphia, PA, United States (U.S. corporation)  
Johns Hopkins University, Baltimore, MD, United States (U.S.  
corporation)  
PI US 6086876 20000711  
AI US 1998-19862 19980206 (9)  
PRAI US 1997-37722P 19970207 (60)  
DT Utility  
FS Granted  
EXNAM Primary Examiner: Kemmerer, Elizabeth; Assistant Examiner: Romeo, David S.  
LREP Akin, Gump, Strauss, Hauer & Feld, L.L.P.  
CLMN Number of Claims: 12  
ECL Exemplary Claim: 1  
DRWN 13 Drawing Figure(s); 18 Drawing Page(s)  
LN.CNT 1487  
CAS INDEXING IS AVAILABLE FOR THIS PATENT.  
AB The invention includes compositions and methods for selective suppression of IL-12 production in a cell. Methods of treating a human having a disease associated with dysregulated IL-12 production are also provided.

L26 ANSWER 15 OF 21 USPATFULL  
AN 2000:50737 USPATFULL  
TI Methods and compositions for modulating responsiveness to corticosteroids  
IN Sekut, Les, Westborough, MA, United States  
Carter, Adam, Newburyport, MA, United States  
Ghayur, Tariq, Grafton, MA, United States  
Banerjee, Subhashis, Shrewsbury, MA, United States  
Tracey, Daniel E., Harvard, MA, United States  
PA BASF Aktiengesellschaft, Rheinland Pfalz, Germany, Federal Republic of (non-U.S. corporation)  
PI US 6054487 20000425  
AI US 1997-820692 19970318 (8)  
DT Utility  
FS Granted  
EXNAM Primary Examiner: Jarvis, William R. A.  
LREP Lahive & Cockfield, LLP  
CLMN Number of Claims: 46  
ECL Exemplary Claim: 1  
DRWN 3 Drawing Figure(s); 3 Drawing Page(s)  
LN.CNT 2404  
CAS INDEXING IS AVAILABLE FOR THIS PATENT.  
AB Method for modulating responsiveness to corticosteroids in a subject are provided. In the method of the invention, an agent which antagonizes a factor that regulates production of IFN-.gamma. in the subject is administered to the subject in combination with a corticosteroid such that responsiveness of the subject to the corticosteroid is modulated as compared to when a corticosteroid alone is administered to the subject. In one embodiment, the agent is an interferon-.gamma. inducing factor (IGIF) antagonist. In another embodiment, the agent is an interleukin-12 (IL-12) antagonist. In a preferred embodiment, the agent is an inhibitor of a caspase family protease, preferably an ICE inhibitor. In another preferred embodiment,

the agent is an anti-**IL-12** monoclonal antibody. Other preferred agents include phosphodiesterase IV inhibitors and beta-2 agonists. The methods of the invention can be used in the treatment of a variety of inflammatory and immunological diseases and disorders. Pharmaceutical compositions comprising an agent which antagonizes a factor that regulates production of IFN-.gamma. in a subject, a corticosteroid and a pharmaceutically acceptable carrier are also provided. A preferred composition comprises an ICE inhibitor, a corticosteroid and a pharmaceutically acceptable carrier.

L26 ANSWER 16 OF 21 USPATFULL  
AN 1999:155952 USPATFULL  
TI Dihomo-seco-cholestanes  
IN Barbier, Pierre, Rixheim, France  
Mohr, Peter, Basel, Switzerland  
Muller, Marc, Saint-Louis, France  
Self, Christopher, West Caldwell, NJ, United States  
PA Hoffmann-La Roche Inc., Nutley, NJ, United States (U.S. corporation)  
PI US 5994569 19991130  
AI US 1998-115188 19980714 (9)  
PRAI EP 1997-112225 19970717  
DT Utility  
FS Granted  
EXNAM Primary Examiner: Dees, Jose' G.; Assistant Examiner: Badio, Barbara  
LREP Johnston, George W., Rocha-Tramaloni, Patricia S., Silverman, Robert A.  
CLMN Number of Claims: 28  
ECL Exemplary Claim: 1  
DRWN No Drawings  
LN.CNT 1220  
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Polyunsaturated 24a,24b-dihomo-9,10-secocholestane derivatives of formula ##STR1## wherein A is a single or double bond,  
B.sup.1 and B.sup.2 are each independently CH.dbd.CH or C.tbd.C,  
T is CH.sub.2 or CH.sub.2 CH.sub.2,  
X is --CH.sub.2 -- or >C.dbd.CH.sub.2,  
R.sup.1 is H, F or OH,  
R.sup.2 and R.sup.3 are each independently lower alkyl or CF.sub.3, or  
C(R.sup.2,R.sup.3) is C.sub.3-6 -cycloalkyl,  
are useful in the treatment or prevention of vitamin D dependent disorders and of **IL-12**-dependent autoimmune diseases, particularly psoriasis, basal cell carcinomas, disorders of keratinization and keratosis, leukemia, osteoporosis, hyperparathyroidism accompanying renal failure, multiple sclerosis, transplant rejection, graft vs. host disease, **rheumatoid arthritis**, insulin-dependent diabetes mellitus, inflammatory bowel disease, septic shock and allergic encephalomyelitis.

L26 ANSWER 17 OF 21 USPATFULL  
AN 1999:75759 USPATFULL  
TI Low affinity human **IL-12** beta2 receptor  
IN Gubler, Ulrich Andreas, Glen Ridge, NJ, United States  
Presky, David Howard, Glen Ridge, NJ, United States  
PA Hoffmann-La Roche Inc., Nutley, NJ, United States (U.S. corporation)  
PI US 5919903 19990706  
AI US 1997-914520 19970819 (8)

RLI Division of Ser. No. US 1996-685118, filed on 23 Jul 1996  
PRAI US 1995-1701P 19950801 (60)  
DT Utility  
FS Granted  
EXNAM Primary Examiner: Draper, Garnette D.  
LREP Johnston, George W., Rocha-Tramaloni, Patricia S., Silverman, Robert A.  
CLMN Number of Claims: 2  
ECL Exemplary Claim: 1  
DRWN No Drawings  
LN.CNT 1531

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB A recombinant human **IL-12** receptor complex produced on the surface of a non-human mammalian cell and free from other human proteins, the complex comprising the betal receptor protein complexed with a beta2 receptor protein, which complex is capable of binding to human **IL-12** with high affinity. A recombinant human **IL-12** beta2 receptor protein produced on the surface of a non-human mammalian cell, free from other human proteins, in its active form. In addition, a non-human mammalian cell having expressed on its surface the recombinant human **IL-12** beta2 receptor protein or the recombinant human **IL-12** receptor complex, which cell proliferates in the presence of human **IL-12**. A non-human mammalian cell having the human **IL-12** beta2 receptor protein or the complex expressed on its surface and which proliferates in response to human **IL-12** is useful for determining whether a given compound inhibits biological activity of human **IL-12** or is an **IL-12** agonist.

L26 ANSWER 18 OF 21 USPATFULL  
AN 1998:160106 USPATFULL  
TI **Antibodies** to receptors for human interleukin-12  
IN Gubler, Ulrich Andreas, Glen Ridge, NJ, United States  
Presky, David Howard, Glen Ridge, NJ, United States  
PA Hoffmann-La Roche Inc., Nutley, NJ, United States (U.S. corporation)  
PI US 5852176 19981222  
AI US 1997-915495 19970820 (8)  
RLI Division of Ser. No. US 1996-685118, filed on 23 Jul 1996  
PRAI US 1995-1701P 19950801 (60)  
DT Utility  
FS Granted  
EXNAM Primary Examiner: Draper, Garnette D.  
LREP Johnston, George W., Rocha-Tramaloni, Patricia S., Silverman, Robert A.  
CLMN Number of Claims: 1  
ECL Exemplary Claim: 1  
DRWN No Drawings  
LN.CNT 1381

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB **Antibodies** to human **IL-12** beta 2 receptor protein or an **IL-12** receptor complex, the complex comprising the betal receptor protein complexed with a beta2 receptor protein, which complex is capable of binding to human **IL-12** with high affinity.

L26 ANSWER 19 OF 21 USPATFULL  
AN 1998:147252 USPATFULL  
TI DNA encoding receptors for the beta-2 chain of human **IL-12**  
IN Gubler, Ulrich Andreas, Glen Ridge, NJ, United States  
Presky, David Howard, Glen Ridge, NJ, United States  
PA Hoffmann-La Roche Inc., Nutley, NJ, United States (U.S. corporation)  
PI US 5840530 19981124

AI US 1996-685118 19960723 (8)  
PRAI US 1995-1701P 19950801 (60)  
US 1996-18674P 19960530 (60)  
DT Utility  
FS Granted  
EXNAM Primary Examiner: Draper, Garnette D.  
LREP Johnston, George W., Rocha-Tramaloni, Patricia S., Silverman, Robert A.  
CLMN Number of Claims: 12  
ECL Exemplary Claim: 1  
DRWN No Drawings  
LN.CNT 1424

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB A recombinant human **IL-12** beta2 receptor protein produced on the surface of a non-human mammalian cell, free from other human proteins, in its active form. In addition, a non-human mammalian cell having expressed on its surface the recombinant human **IL-12** beta2 receptor protein, which cell proliferates in the presence of human **IL-12**. A non-human mammalian cell having the human **IL-12** beta2 receptor protein on its surface and which proliferates in response to human **IL-12** is useful for determining whether a given compound inhibits biological activity of human **IL-12** or is an **IL-12** agonist.

L26 ANSWER 20 OF 21 USPATFULL  
AN 1998:135151 USPATFULL  
TI Human receptor for interleukin-12  
IN Chua, Anne On, Wayne, NJ, United States  
Gubler, Ulrich Andreas, Glen Ridge, NJ, United States  
PA Hoffmann-La Roche Inc., Nutley, NJ, United States (U.S. corporation)  
PI US 5831007 19981103  
AI US 1995-419652 19950411 (8)  
RLI Division of Ser. No. US 1994-248532, filed on 31 May 1994, now patented, Pat. No. US 5536657 which is a continuation-in-part of Ser. No. US 1993-94713, filed on 19 Jul 1993, now abandoned

DT Utility  
FS Granted  
EXNAM Primary Examiner: Ulm, John  
LREP Johnston, George W., Epstein, William H., Bucholz, Briana C.  
CLMN Number of Claims: 10  
ECL Exemplary Claim: 1  
DRWN 35 Drawing Figure(s); 26 Drawing Page(s)  
LN.CNT 1937

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB This invention relates to substantially pure Interleukin-12 receptor cDNAs and protein and uses therefore. The Interleukin-12 receptor is shown to be a member of the cytokine receptor superfamily and has a high homology to human gp130.

L26 ANSWER 21 OF 21 USPATFULL  
AN 96:63048 USPATFULL  
TI Recombinant DNA encoding human receptor for interleukin-12  
IN Chua, Anne O., Wayne, NJ, United States  
Gubler, Ulrich A., Glen Ridge, NJ, United States  
PA Hoffmann-La Roche Inc., Nutley, NJ, United States (U.S. corporation)  
PI US 5536657 19960716  
AI US 1994-248532 19940531 (8)  
RLI Continuation-in-part of Ser. No. US 1993-94713, filed on 19 Jul 1993, now abandoned  
DT Utility  
FS Granted  
EXNAM Primary Examiner: Ulm, John

LREP Gould, George M., Johnston, George W., Kass, Alan P.  
CLMN Number of Claims: 10  
ECL Exemplary Claim: 1  
DRWN 34 Drawing Figure(s); 25 Drawing Page(s)  
LN.CNT 1755  
CAS INDEXING IS AVAILABLE FOR THIS PATENT.  
AB This invention relates to substantially pure Interleukin-12 receptor cDNAs and protein and uses therefore. The Interleukin-12 receptor is shown to be a member of the cytokine receptor superfamily and has a high homology to human gp130.

=> d clm 12 15 18

L26 ANSWER 12 OF 21 USPATFULL  
CLM What is claimed is:  
1. Pharmaceutical treatment material comprising in combination (i) **IL-12**, or a functional homologue thereof, for use as an adjuvant, and (ii) a protein consisting essentially of at least one antigenic portion of a papillomavirus protein, wherein (a) the papillomavirus is selected from the group consisting of HPV types 6, 11, 16 and 18, and (b) the papillomavirus protein is selected from the group consisting of E6, E7, L1 and L2 proteins.  
2. The pharmaceutical treatment material according to claim 1, wherein the papillomavirus protein is selected from the group consisting of E7 and L2.  
3. The pharmaceutical treatment material according to claim 1, wherein the vaccine adjuvant is **IL-12** or a protein that differs from **IL-12** by one or more conservative amino acid substitutions and which retains IL12 activity.  
4. The pharmaceutical treatment material according to claim 1, wherein the vaccine adjuvant is **IL-12**.  
5. Pharmaceutical treatment material comprising in combination (i) **IL-12**, or a functional homologue thereof, for use as an adjuvant, and (ii) a nucleic acid molecule encoding a protein consisting essentially of at least one antigenic portion of a papillomavirus protein, wherein (a) the papillomavirus is selected from the group consisting of HPV types 6, 11, 16 and 18, and (b) the papillomavirus protein is selected from the group consisting of E6, E7, L1 and L2 proteins.  
6. The pharmaceutical treatment material according to claim 5, wherein the papillomavirus protein is selected from the group consisting of E7 and L2.  
7. The pharmaceutical treatment material according to claim 5, wherein the vaccine adjuvant is **IL-12** or a protein that differs from **IL-12** by one or more conservative amino acid substitutions and which retains IL12 activity.  
8. The pharmaceutical treatment material according to claim 5, wherein the vaccine adjuvant is **IL-12**.  
9. The pharmaceutical treatment material of claim 1, wherein the papillomavirus is HPV type 16.  
10. The pharmaceutical treatment material of claim 1, wherein the papillomavirus protein is selected from the group consisting of E6 and

E7.

11. The pharmaceutical treatment material of claim 1, wherein the papillomavirus protein is E7.
12. The pharmaceutical treatment material of claim 1, wherein the papillomavirus virus is HPV type 16 and the papillomavirus protein is E7.
13. The pharmaceutical treatment material of claim 5, wherein the papillomavirus virus is HPV type 16 and the papillomavirus protein is E7.

L26 ANSWER 15 OF 21 USPATFULL

CLM What is claimed is:

1. A method for modulating responsiveness to a corticosteroid in a subject, comprising administering to the subject suffering from a condition normally responsive to corticosteroid therapy: an interleukin-1 .beta. converting enzyme (ICE) inhibitor being administered at a dosage and by a route sufficient to inhibit production of IFN-.gamma. in the subject; and a corticosteroid, such that responsiveness of the subject to the corticosteroid is modulated as compared to when a corticosteroid alone is administered to the subject.
2. The method of claim 1, wherein the ICE inhibitor is an IFN-.gamma. inducing factor (IGIF) **antagonist**, the ICE inhibitor being administered at a dosage and by a route sufficient to inhibit IGIF activity in the subject.
3. The method of claim 1, wherein the corticosteroid is selected from the group consisting of cortisone, hydrocortisone, beclomethasone, flunisolide, prednisone, prednisolone, methylprednisolone, triamcinolone, deflazacort, betamethasone and dexamethasone.
4. The method of claim 1, wherein the subject is suffering from septic shock.
5. The method of claim 1, wherein the subject is suffering from Crohn's disease.
6. The method of claim 1, wherein the subject is suffering from asthma.
7. The method of claim 1, wherein the subject is suffering from graft versus host disease or transplant rejection.
8. The method of claim 1, wherein the subject is suffering from an autoimmune disease or disorder.
9. The method of claim 1, wherein the subject is suffering from an immunoinflammatory disease or disorder selected from the group consisting of asthma, adult respiratory distress syndrome, systemic lupus erythematosus, inflammatory bowel disease, Crohn's disease, ulcerative colitis, multiple sclerosis, insulin-dependent diabetes mellitus, autoimmune arthritis, **rheumatoid arthritis**, juvenile **rheumatoid arthritis**, psoriatic arthritis, inflammatory pulmonary syndrome, pemphigus vulgaris, idiopathic thrombocytopenic purpura, autoimmune meningitis, myasthenia gravis, autoimmune thyroiditis, dermatitis, atopic dermatitis, eczematous dermatitis, psoriasis, Sjogren's Syndrome, keratoconjunctivitis sicca secondary to Sjogren's Syndrome, alopecia areata, allergic responses due to arthropod bite reactions, aphthous

ulcer, iritis, conjunctivitis, keratoconjunctivitis, cutaneous lupus erythematosus, scleroderma, vaginitis, proctitis, drug eruptions, Stevens-Johnson syndrome, leprosy reversal reactions, erythema nodosum leprosum, autoimmune uveitis, allergic encephalomyelitis, aplastic anemia, pure red cell anemia, idiopathic thrombocytopenia, polychondritis, Wegener's granulomatosis, chronic active hepatitis, Graves ophthalmopathy, primary biliary cirrhosis, uveitis posterior and interstitial lung fibrosis.

10. The method of claim 1, wherein the subject is suffering from an acute inflammatory disorder.
11. The method of claim 1, wherein the subject is suffering from a chronic inflammatory disorder.
12. The method of claim 1, wherein the ICE inhibitor and corticosteroid are administered such that steroid resistance in the subject is reversed, as compared to when a corticosteroid alone is administered to the subject.
13. The method of claim 1, wherein the ICE inhibitor and corticosteroid are administered such that steroid sensitivity in the subject is increased, as compared to when a corticosteroid alone is administered to the subject.
14. The method of claim 1, wherein the ICE inhibitor and the corticosteroid are administered to the subject according to a schedule that reduces the dosage of the corticosteroid over time and a method ameliorates a steroid rebound effect associated with administration of reduced dosages of the corticosteroid.
15. A method for modulating responsiveness to corticosteroids in a subject, comprising administering to the subject suffering from a condition normally responsive to corticosteroid therapy, an interleukin-1 .beta. converting enzyme (ICE) inhibitor; and a corticosteroid, such that responsiveness of the subject to the corticosteroid is modulated as compared to when a corticosteroid alone is administered to the subject.
16. The method of claim 15, wherein the corticosteroid is selected from the group consisting of cortisone, hydrocortisone, beclomethasone, flunisolide, prednisone, prednisolone, methylprednisolone, triamcinolone, deflazacort, betamethasone and dexamethasone.
17. The method of claim 15, wherein the subject is suffering from septic shock.
18. The method of claim 15, wherein the subject is suffering from Crohn's disease.
19. The method of claim 15, wherein the subject is suffering from asthma.
20. The method of claim 15, wherein the subject is suffering from graft versus host disease or transplant rejection.
21. The method of claim 15, wherein the subject is suffering from an autoimmune disease or disorder.
22. The method of claim 15, wherein the subject is suffering from an immunoinflammatory disease or disorder selected from the group consisting of asthma, adult respiratory distress syndrome, systemic

*lupus erythematosus, inflammatory bowel disease, Crohn's disease, ulcerative colitis, multiple sclerosis, insulin-dependent diabetes mellitus, autoimmune arthritis, rheumatoid arthritis, juvenile rheumatoid arthritis, psoriatic arthritis, inflammatory pulmonary syndrome, pemphigus vulgaris, idiopathic thrombocytopenic purpura, autoimmune meningitis, myasthenia gravis, autoimmune thyroiditis, dermatitis, atopic dermatitis, eczematous dermatitis, psoriasis, Sjogren's Syndrome, keratoconjunctivitis sicca secondary to Sjogren's Syndrome, alopecia areata, allergic responses due to arthropod bite reactions, aphthous ulcer, iritis, conjunctivitis, keratoconjunctivitis, cutaneous lupus erythematosus, scleroderma, vaginitis, proctitis, drug eruptions, Stevens-Johnson syndrome, leprosy reversal reactions, erythema nodosum leprosum, autoimmune uveitis, allergic encephalomyelitis, aplastic anemia, pure red cell anemia, idiopathic thrombocytopenia, polychondritis, Wegener's granulomatosis, chronic active hepatitis, Graves ophthalmopathy, primary biliary cirrhosis, uveitis posterior and interstitial lung fibrosis.*

23. The method of claim 15, wherein the subject is suffering from an acute inflammatory disorder.
24. The method of claim 15, wherein the subject is suffering from a chronic inflammatory disorder.
25. The method of claim 24, wherein the ICE inhibitor and the corticosteroid are administered such that steroid resistance in the subject is reversed, as compared to when a corticosteroid alone is administered to the subject.
26. The method of claim 24, wherein the ICE inhibitor and the corticosteroid are administered such that steroid sensitivity in the subject is increased, as compared to when a corticosteroid alone is administered to the subject.
27. The method of claim 24, wherein the ICE inhibitor and the corticosteroid are administered to the subject according to a schedule that reduces the dosage of the corticosteroid over time and the method ameliorates a steroid rebound effect associated with administration of reduced dosages of the corticosteroid.
28. A method for modulating responsiveness to a corticosteroid in a subject, comprising: selecting a subject in need of modulation of responsiveness to a corticosteroid, wherein the subject suffers from a condition normally responsive to corticosteroid therapy; and administering to the subject an interleukin-1 beta. converting enzyme (ICE) inhibitor which antagonizes a factor that regulates production of interferon (IFN-.gamma.) in the subject, the ICE inhibitor being administered at a dosage and by a route sufficient to inhibit production of IFN-.gamma. in the subject, such that responsiveness of the subject to a corticosteroid is modulated as compared to when a corticosteroid alone is administered to the subject.
29. The method of claim 28, wherein the subject is resistant to a corticosteroid prior to administration of the ICE inhibitor.
30. The method of claim 28, wherein the subject is responsive to a corticosteroid prior to administration of the ICE inhibitor but exhibits increased sensitivity to the corticosteroid after administration of the ICE inhibitor.
31. The method of claim 28, wherein treatment of the subject with a

corticosteroid is to be stopped and administration of the ICE inhibitor ameliorates a steroid rebound effect in the subject.

32. The method of claim 28, wherein the ICE inhibitor is an IFN-.gamma. inducing factor (IGIF) antagonist, the ICF inhibitor being administered at a dosage and by a route sufficient to inhibit IGIF activity in the subject.

33. A method for modulating responsiveness to corticosteroids in a subject comprising administering to the subject suffering from a condition normally responsive to corticosteroid therapy: an interleukin-1.beta. converting enzyme (ICE) inhibitor compound having the structure of Formula I: ##STR6## wherein R.sup.1 is hydrogen, C.sub.1 -C.sub.6 alkyl, or benzyl; R.sup.2 is --CHO, --COR.sup.a, or --CN; each R.sup.a is independently hydrogen or C.sub.1 -C.sub.6 alkyl; X is a bond, CH.sub.2, CHR.sup.5, NH, NR.sup.5, or O; R.sup.3 is aryl, substituted-aryl, heteroaryl, substituted-heteroaryl, cycloalkyl, substituted-cycloalkyl, heterocycle, or substituted-heterocycle; Y is absent, NR.sup.5, CO, S, O, SO.sub.2, --O(CHR.sup.5).sub.n --, CHR.sup.5, NR.sup.5 CO, NC(O)R.sup.5, CONR.sup.5, OCHR.sup.5, CHR.sup.5 O, SCHR.sup.5, CHR.sup.5 S, SO.sub.2 NR.sup.5, C.sub.1 -C.sub.6 alkyl, NR.sup.5 SO.sub.2, CH.sub.2 CHR.sup.5, CHR.sup.5 CH.sub.2, COCH.sub.2, or CH.sub.2 CO; R.sup.4 is absent, aryl, substituted-aryl, C.sub.1 -C.sub.8 alkyl, heteroaryl, substituted-heteroaryl, cycloalkyl, C.sub.1 -C.sub.6 alkyl, substituted-cycloalkyl, heterocycloalkyl, or substituted-heterocycloalkyl; each R.sup.5 is independently hydrogen, C.sub.1 -C.sub.6 alkyl, aryl, -(CH.sub.2).sub.n aryl, or -(CH.sub.2).sub.n cycloalkyl; each n is independently 0 to 5, m is 1 or 2, and the pharmaceutically acceptable salts, esters, amides, and prodrugs thereof; and a corticosteroid, such that responsiveness of the subject to the corticosteroid is modulated as compared to when a corticosteroid alone is administered to the subject.

34. A method for modulating responsiveness to a corticosteroid in a subject, comprising: selecting a subject in need of modulation of responsiveness to a corticosteroid, wherein the subject suffers from a condition normally responsive to corticosteroid therapy; and administering to the subject an interleukin-1.beta. converting enzyme (ICE) inhibitor compound having The structure of Formula I: ##STR7## wherein R.sup.1 is hydrogen, C.sub.1 -C.sub.6 alkyl, or benzyl; R.sup.2 is --CHO, --COR.sup.a, or --CN; each R.sup.a is independently hydrogen or C.sub.1 -C.sub.6 alkyl; X is a bond, CH.sub.2, CHR.sup.5, NH, NR.sup.5, or O; R.sup.3 is aryl, substituted-aryl, heteroaryl, substituted-heteroaryl, cycloalkyl, substituted-cycloalkyl, heterocycle, or substituted-heterocycle; Y is absent, NR.sup.5, CO, S, O, SO.sub.2, --O(CHR.sup.5).sub.n --, CHR5, NR.sup.5 CO, NC(O)R.sup.5, CONR.sup.5, OCHR.sup.5, CHR.sup.5 O, SCHR.sup.5, CHR.sup.5 S, SO.sub.2 NR.sup.5, C.sub.1 -C.sub.6 alkyl, NR.sup.5 SO.sub.2, CH.sub.2 CHR.sup.5, CHR.sup.5 CH.sub.2, COCH.sub.2, or CH.sub.2 CO; R.sup.4 is absent, aryl, substituted-aryl, C.sub.1 -C.sub.8 alkyl, heteroaryl, substituted-heteroaryl, cycloalkyl, C.sub.1 -C.sub.6 alkyl, substituted-cycloalkyl, heterocycloalkyl, or substituted-heterocycloalkyl; each R.sup.5 is independently hydrogen, C.sub.1 -C.sub.6 alkyl, aryl, -(CH.sub.2).sub.n aryl, or -(CH.sub.2).sub.n cycloalkyl; each n is independently 0 to 5, m is 1 or 2, and the pharmaceutically acceptable salts, esters, amides, and prodrugs thereof, the compound being administered at a dosage and by a route sufficient to inhibit production of IFN-.gamma. in the subject, such that responsiveness of the subject to a corticosteroid is modulated as compared to when a corticosteroid alone is administered to the subject.

35. A method of claim 9, wherein the subject is suffering from an

immunoinflammatory disease or disorder selected from the group consisting of pemphigus vulgaris, dermatitis, atopic dermatitis, eczematous dermatitis, psoriasis, alopecia areata, allergic responses due to arthropod bite reactions, cutaneous lupus erythematosus, scleroderma, vaginitis, drug eruptions, Stevens-Johnson syndrome, leprosy reversal reactions, and erythema nodosum leprosum.

36. A method of claim 9, wherein the subject is suffering from an immunoinflammatory disease or disorder selected from the group consisting of multiple sclerosis, autoimmune arthritis, **rheumatoid arthritis**, juvenile **rheumatoid arthritis**, psoriatic arthritis, autoimmune meningitis, myasthenia gravis and allergic encephalomyelitis.

37. A method of claim 9, wherein the subject is suffering from an immunoinflammatory disease or disorder selected from the group consisting of systemic lupus erythematosus, inflammatory bowel disease, Crohn's disease, ulcerative colitis, insulin-dependent diabetes mellitus, aphthous ulcer, proctitis, Wegener's granulomatosis, chronic active hepatitis, and primary biliary cirrhosis.

38. A method of claim 9, wherein the subject is suffering from an immunoinflammatory disease or disorder selected from the group consisting of iritis, conjunctivitis, keratoconjunctivitis, autoimmune uveitis, Graves ophthalmopathy, and uveitis posterior.

39. A method of claim 9, wherein the subject is suffering from an immunoinflammatory disease or disorder selected from the group consisting of idiopathic thrombocytopenic purpura, autoimmune thyroiditis, Sjogren's Syndrome, keratoconjunctivitis sicca secondary to Sjogren's Syndrome, aplastic anemia, pure red cell anemia, idiopathic thrombocytopenia, and polychondritis.

40. The method of claim 9, wherein the subject is suffering from an immunoinflammatory disease or disorder selected from the group consisting of asthma, adult respiratory distress syndrome, inflammatory pulmonary syndrome, and interstitial lung fibrosis.

41. A method of claim 22, wherein the subject is suffering from an immunoinflammatory disease or disorder selected from the group consisting of pemphigus vulgaris, dermatitis, atopic dermatitis, eczematous dermatitis, psoriasis, alopecia areata, allergic responses due to arthropod bite reactions, cutaneous lupus erythematosus, scleroderma, vaginitis, drug eruptions, Stevens-Johnson syndrome, leprosy reversal reactions, and erythema nodosum leprosum.

42. A method of claim 22, wherein the subject is suffering from an immunoinflammatory disease or disorder selected from the group consisting of mutiple sclerosis, autoimmune arthritis, **rheumatoid arthritis**, juvenile **rheumatoid arthritis**, psoriatic arthritis, autoimmune meningitis, myasthenia gravis and allergic encephalomyelitis.

43. A method of claim 22, wherein the subject is suffering from an innumoinflammatory disease or disorder selected from the group consisting of systemic lupus erythematosus, inflammatory bowel disease, Crohn's disease, ulcerative colitis, insulin-dependent diabetes mellitus, aphthous ulcer, procitis, Wegener's granulomatosis, chronic active hepatitis, and primary biliary cirrhosis.

44. A method of claim 22, wherein the subject is suffering from an inflammatory disease or disorder selected from the group consisting of

ireitis, conjunctivitis, keratoconjunctivitis, autoimmune eyeitis, Graves ophthalmopathy, and uveitis posterior.

45. A method of claim 22, wherein the subject is suffering from an immunoinflammatory disease or disorder selected from the group consisting of idiopathic thrombocytopenic purpura, autoimmune thyroiditis, Sjogren's Syndrome, keratoconjunctivitis sicca secondary to Sjogren's Syndrome, aplastic anemia, pure red cell anemia, idiopathic thrombocytopenia, and polychondritis.

46. The method of claim 22, wherein the subject is suffering from an immunoinflammatory disease or disorder selected from the group consisting of asthma, adult respiratory distress syndrome, inflammatory pulmonary syndrome, and interstitial lung fibrosis.

L26 ANSWER 18 OF 21 USPATFULL

CLM What is claimed is:

1. An **antibody** directed against a interleukin-12 (**IL-12**) beta2 receptor protein which protein (a) has low binding affinity for human **IL-12**, and (b) when complexed with a human **IL-12** betal receptor protein forms a complex having high binding affinity to human **IL-12**.